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VOLUME 32 NUMBER 5

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PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO, ILLINOIS. ANNUAL SUBSCRIPTION, $8.00

Entered as Second-Class Matter, July 10, 1925, at the Postoffice at Chicago, Illinois, Under the Act of March 3, 1879.

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ARCHIVES OF SURGERY

VOLUME 32 MAY 1936 NUMBER 5

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EVAPORATION OF WATER FROM SUPERFICIAL BURNS

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ROCHESTER, N. Y.

The object of the experiments was to answer the questions: Does evaporation from burned surfaces account for a significant portion of the dehydration of the body that is believed to be present after accidental burns? Does the treatment of these surfaces with tannic acid materially modify this evaporation?

METHOD

Apparatus.—A glass capsule was fitted with inlet and outlet tubes opposite one another. It enclosed an area of skin of 22 sq. cm. A thermocouple on the end of a piece of bakelite tubing was held inside the capsule, so that it was pressed lightly against the surface of the skin at all times. Wires to a cold junction and to a galvanometer passed through the tubing and the sealed top of the capsule. The capsule was sealed air-tight to the cutaneous surface with petrolatum. A stream of air was measured by passage through a dry meter, was dried by passage through a spiral washing-bottle containing concentrated sulfuric acid and was drawn through the capsule by suction. Tests were made for leaks around the capsule by pinching momentarily the tubing that led the air into the capsule.

The moisture acquired by the air while in the capsule was collected in weighed flasks of concentrated sulfuric acid during ten minute periods. The flow of air was approximately constant at 0.5 liter per minute, but variations in the flow from 0.2 to 3 liters per minute produced no significant changes in the rate of evaporation. Rates of evaporation were calculated as milligrams of water per minute per square centimeter of surface.

Blank measurements with a dry glass plate in place of the cutaneous surface showed an average gain by the collecting flasks of 0.5 mg. in ten minutes. Since the average amount of moisture picked up from the skin in any one determination was 10 mg., the error was 5 per cent.

Cutaneous temperatures were determined with the thermocouple from the readings of a galvanometer and of a mercury thermometer at the cold junction, which was immersed in a thermos bottle of water at approximately 26 C.

Conditions.—The measurements were carried out in a room in which the temperature was controlled within ± 1 C. and the humidity within ± 8 per cent, with a mean temperature of 26 C. and a mean relative humidity of 50 per cent.

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This investigation was supported in part by the Fluid Research Fund of the Rockefeller Foundation. The patients studied were in the Strong Memorial Hospital and the Rochester Municipal Hospital.

Subjects were lightly clothed and were always in the room for thirty minutes before the measurements were started in order to adjust themselves to the environmental temperature and humidity. To remove any moisture already on the skin or in the tubing, the dry air was passed for twenty minutes before collections were made. Several successive collections were made from each area studied, and whenever possible determinations were made not only from the burned area but also from a corresponding normal area. The determinations were repeated daily at the same hour until the burned area seemed to be normal again.

The rates of evaporation from the burned and normal areas must be considered relative rather than representative of the actual rates that would prevail outside the laboratory, since the air used in the experiment was dry and moving. Vasti [1] found that both humidity and cutaneous temperature within certain limits affect the local evaporation from the skin. Similarly, the temperatures of the skin measured inside the capsule are not accurately characteristic of the normal surface; but with uniform rates of air flow the values have relative significance.

Control Procedures.—Control measurements made on normal persons during the present study showed a maximum difference of 20.8 per cent and an average difference of 8.4 per cent in 100 pairs of successive determinations on many different subjects. Variations in different parts of the body of the same person were considerable, the forehead having the greatest rate of evaporation, 0.0686 mg. per square centimeter per minute, and the extensor surface of the forearm having the least, 0.0343 mg. per square centimeter per minute. The mean for the entire body was 0.0454 mg. per square centimeter per minute, or 27 Gm. per square meter per hour. The evaporation was greater from flexor surfaces, owing possibly to the fact that more sweat glands are present per unit of area on flexor surfaces and that the texture of the skin is finer. The greatest variation on different days in the rate of evaporation from corresponding areas was 43 per cent, and the mean difference was 11.8 per cent.

In some cases the presence or absence of activity of the sweat glands was determined microscopically in the manner suggested by Jürgensen.[2] A drop of liquid petrolatum containing 2 per cent methyl red was placed on the skin and observed under a microscope. If the sweat glands were active, the brighter droplets of sweat as they formed could be observed against the darker background of the red oil.

Mild first degree burns were produced on normal volunteers by exposure to ultraviolet radiation from a quartz mercury vapor arc lamp. The rates of evaporation and the cutaneous temperatures were followed just as in the clinical cases. Like determinations were made following the application of tannic acid to normal skin.

RESULTS

Rates of Evaporation.—These were measured at the same time of day for many successive days. That the differences from normal were consistent is shown by chart 1, which represents data on one of six subjects in whom first and second degree burns were studied simultaneously. Chart 2 shows a composite graph of the rates of evaporation in eight cases of second degree burn, before and after the application of tannic acid.

In the majority of cases, before the application of tannic acid the rates of evaporation were found to be high, sometimes as much as 400 to 500 per cent as

1. Vasti, A.: The Insensible Water Loss Through the Skin, Am. J. Physiol. **102**:60, 1933.

2. Jürgensen, E.: Mikrobeobachtungen der Schweissekretion der Haut des Menschen unter Kontrast-Färbung, Deutsches Arch. f. klin. Med. **144**:193, 1924.

compared with those from a corresponding normal area. Following the application of tannic acid the rates of evaporation fell rapidly, reaching a subnormally low level when the area was fully tanned. In case 8, the burn had been caused by boiling paraffin, while in cases 6 and 11 petrolatum and ointment had been applied prior to the patient's admission to the hospital. Traces of these substances remaining on the skin probably inhibited the evaporation and caused the comparatively low initial rates observed in these particular cases.

The rates of evaporation remained at the subnormal levels for five or six days and then began to rise gradually. In most cases after the removal of the eschars the rates rose markedly to high levels and then dropped gradually to constant rates

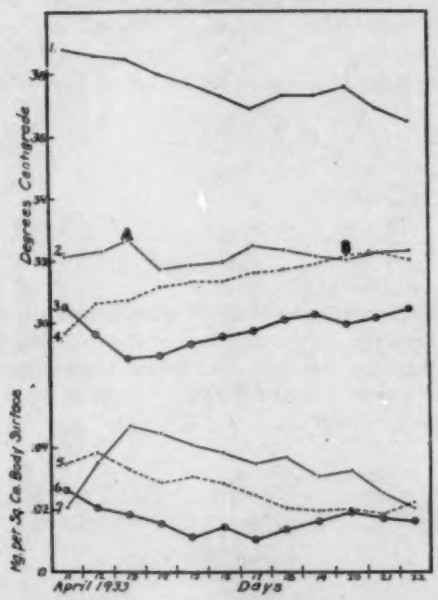

Fig. 1.—Temperatures and rates of evaporation in a patient burned on the back on April 11. Rates of evaporation are expressed in milligrams of moisture per minute per square centimeter of body surface. Line 1 indicates the rectal temperatures; line 2 indicates the temperatures in a corresponding normal area; line 3 indicates the temperatures in an area of second degree burn, and line 4 indicates those in an area of first degree burn. Line 5 indicates the rates of evaporation from an area of first degree burn; line 6 indicates the rates of evaporation from an area of second degree burn, and line 7 indicates those from a corresponding normal area. Treatment with tannic acid was stopped at *A*, and the eschars were removed at *B*.

slightly below normal. In all cases the evaporation from an area completely regenerated after a second degree burn was less than that from the corresponding normal area.

Chart 3 is a composite graph of the rates of evaporation in six cases of first degree burn. In all, the initial rates were abnormally high and gradually decreased

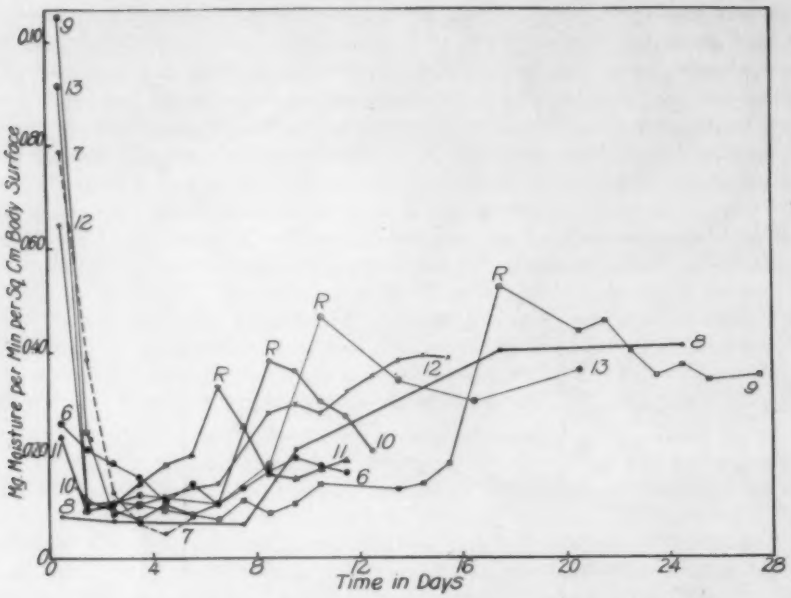

Fig. 2.—Rates of evaporation of water from areas of second degree burns. In each case the burn occurred on the first day of the experiment, and the measurements recorded on that day were obtained before treatment with tannic acid was begun. Tanning was usually completed before the second day's measurement. At *R* the eschars were removed.

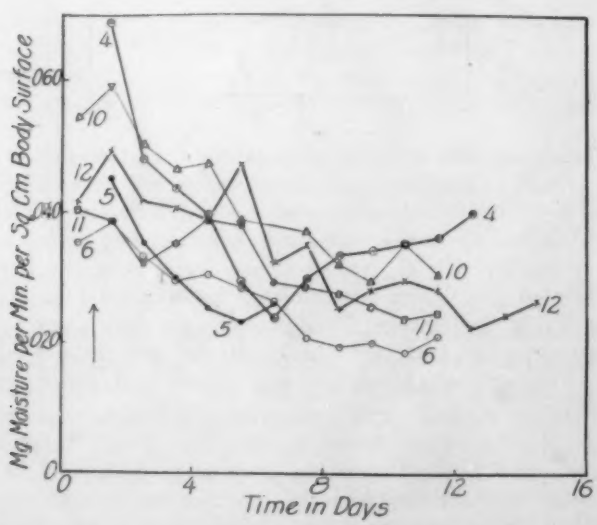

Fig. 3.—Rates of evaporation from areas of first degree burns. In each case the burn occurred on the first day of the experiment.

to a level equal to that of the corresponding normal area. Tanning was never effective over an area of first degree burn because insufficient amounts of broken-down necrotic epithelial tissue were present to form a coagulum with the tannic acid.

The rates of evaporation from normal areas rose somewhat during the first few days, usually reaching a peak on the third or fourth day. This rise might be considered a gradual return to normal after an initial disturbance produced by the burn, or, in some cases, it might be due to the accompanying rise in general body temperature which was observed.

Temperatures.—The surface temperatures in diverse areas of skin varied directly as the rates of evaporation (chart 1). In the tanned areas the low cutaneous temperatures that were found were probably due to the thickness and insulating properties of the tannic acid covering. In areas of first degree burns the cutaneous

Rates of Evaporation and Surface Temperatures in Injured Areas of Patients Suffering from Accidental Burns

Case number	4	5	6	7	8	9	10	11	12	13
Second degree burns										
Evaporation (mg. per sq. cm. per min.)										
Initial	.....	.....	0.027	0.078	0.008	0.105	.....	0.024	0.065	0.091
Minimum after treatment with tannic acid	0.005	0.006	0.010	0.005	0.007	0.008	0.008	0.007	0.010	0.009
After recovery	.....	.....	0.019	.....	0.039	0.035	0.021	0.019	0.039	0.036
Corresponding normal area	0.047	0.072	0.030	.....	0.041	0.040	0.029	0.025	.....	0.033
Cutaneous temperature (C.)										
Initial	....	....	30.5	....	....	....	....	32.2	29.8	33.8
After treatment with tannic acid	26.4	24.8	29.0	....	....	....	29.8	31.2	32.4	29.9
After recovery	30.5	....	30.4	....	....	33.3	32.0	31.3	33.3	31.7
Corresponding normal area	29.0	27.9	32.1	....	....	32.3	32.6	32.5	....	31.6
First degree burns										
Evaporation (mg. per sq. cm. per min.)										
Initial	0.069	0.045	0.034	.....	.....	.....	0.054	0.040	0.042	
After recovery	0.036	.....	0.020	.....	.....	.....	0.030	0.025	0.026	
Cutaneous temperature (C.)										
Initial	32.5	31.0	29.6	....	....	....	34.6	32.4	31.1	
After recovery	29.8	....	32.3	....	....	....	32.0	32.1	32.2	

temperatures were higher than normal, corresponding to the higher rates of evaporation. The daily fluctuations in cutaneous temperatures did not usually correspond, however, to the daily variations in rate of evaporation.

The table shows the average values for cutaneous temperature and rate of evaporation in all but the first three cases studied. These three were not as adequately controlled as were the later experiments, and the data are therefore omitted. Of the thirteen subjects, five were children from 2 to 5 years of age.

Sweat Glands.—The activity of sweat glands was tested in several cases. Repeated microscopic examination of the healed areas of second degree burns failed to reveal activity of the sweat glands, but the sweat glands are not actually destroyed in second degree burns.[3] Examination of normal areas and of healed areas of first degree burns showed active sweat glands in all cases. Richardson[4]

3. Pack, G. T., and Davis, A. H.: Burns, Philadelphia, J. B. Lippincott Company, 1930, p. 23.

4. Richardson, H. B.: The Effect of the Absence of Sweat Glands on the Elimination of Water from the Skin and Lungs, J. Biol. Chem. **67**:397, 1926.

found that in a person with congenital absence of sweat glands the insensible loss of moisture at rest was as great as that of a normal person.

Frost-Bite.—This was studied in the same manner in two cases in which tannic acid therapy was applied. In one case both feet were affected; in the other one foot served as a normal control. The rates of evaporation and the cutaneous temperatures were very high at first, fell markedly after the application of tannic acid and then rose gradually to normal. The results were precisely similar to those in cases of second degree burns.

Burns Caused by Ultraviolet Radiation.—Burns caused by ultraviolet radiation of the skin were studied in two normal persons on four occasions. The rates of evaporation were always abnormally low immediately after the exposure, but a few hours later they began to rise (chart 4), and by the following day they were (in experiment 4) as much as 200 to 300 per cent above the values for the corresponding normal areas. As the area healed, there was a gradual return to the normal level that had been observed before the treatment. When the dead skin peeled off,

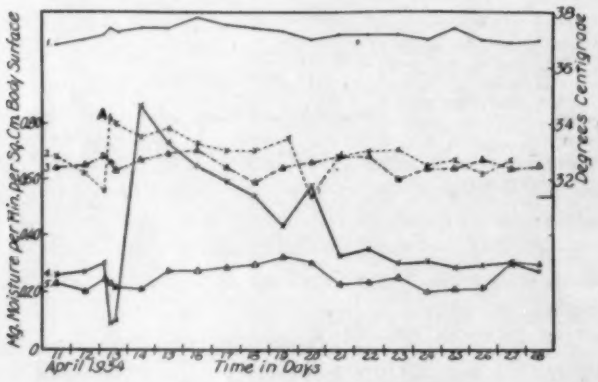

Fig. 4 (experiment 4, subject G. S. M.).—Temperatures and rates of evaporation of water from the flexor surfaces of both forearms, the left being burned once with ultraviolet radiation. Line 1 indicates the rectal temperature of the subject; line 2 indicates the temperatures of the irradiated area, and line 3 indicates those of the corresponding normal area. Line 4 indicates the rate of evaporation from the irradiated area, and line 5 indicates that from the corresponding normal area. Irradiation occurred at *A*.

the rate of evaporation rose markedly for a day or two and then fell again. The cutaneous temperature in general rose and fell with the rate of evaporation. Tannic acid did not form a coagulum over these areas and seemed to have no effect on the rate of evaporation, such areas resembling, therefore, first degree accidental burns.

The temporary inhibition of evaporation immediately following exposure, which was found in all four experiments, might have been due to some direct effect of the radiation on cellular activities or on the superficial blood vessels. This decrease in the rate of evaporation may be a contributing cause of the fever associated with sunstroke, since it indicates that loss of heat is retarded, at least in part.

Normal Skin.—On normal skin no coagulum formed when the skin was treated with tannic acid, and the application of tannic acid caused no appreciable change in the rate of evaporation. Attempts to form a coagulum on normal skin by first applying egg albumin were likewise unsuccessful. Hence it was not possible to ascertain the effect of tannic acid on the skin on other than injured surfaces.

COMMENT

The amount of fluid lost from the body owing to an accidental burn may now be calculated; for this purpose the most extreme of the burns studied is chosen. In case 9, a woman 40 years old, weighing 91 Kg. and having a calculated surface area of 1.98 square meters, had a second degree burn involving one third of the surface of the body.[5] The initial rates of evaporation from normal, burned and tanned areas were 0.041, 0.105 and 0.008 mg. per square centimeter per minute, respectively. From these figures it is calculated that the twenty-four hour loss of water would normally have been 1.29 per cent of the total body weight, but since one third of the surface of the body was losing water at a rate approximately 2½ times normal, the twenty-four hour loss was increased to 2.05 per cent of the total weight. After the application of tannic acid to the burned area, the twenty-four hour loss was reduced to 1.16 per cent of the total weight of the body. A woman of this weight would have an approximate volume of circulating blood of 7,000 cc. and a normal rate of evaporation of 1,175 Gm. in twenty-four hours. Underhill[6] expressed the belief that the fluid lost from burned areas is essentially blood plasma; from this it would follow that, since the burn involved one third of the surface of the body and since the rate of evaporation increased to 1,835 Gm. in twenty-four hours, the blood volume might be decreased at most by the difference between the normal rate and the rate after injury, or 660 Gm.

It may be concluded, therefore, that evaporation has a negligible effect in producing dehydration after a severe burn. Even in children evaporation alone could not be important. Increased concentration of the blood, such as was found by Underhill[7] and others, can hardly be due ordinarily to loss of water from the body. This is confirmed by the constancy of the body weight during convalescence from burns. Hence water that leaves the blood probably remains in the body, in

5. Berkow, S. G.: A Method of Estimating the Extensiveness of Lesions (Burns and Scalds) Based on Surface Area Proportions, Arch. Surg. **8**:138 (Jan.) 1924.

6. Underhill, F. P.; Kapsinow, R., and Fisk, M. E.: Changes in Capillary Permeability Induced by a Superficial Burn, Am. J. Physiol. **95**:315, 1930.

7. Underhill, F. P.; Carrington, G. L.; Kapsinow, R., and Pack, G. T.: Blood Concentration Changes in Extensive Superficial Burns, and Their Significance for Systemic Treatment, Arch. Int. Med. **32**:31 (July) 1923. Underhill, F. P.: The Significance of Anhydremia in Extensive Superficial Burns, J. A. M. A. **95**:852 (Sept. 20) 1930.

the spaces in various tissues.[8] Restoration of the normal distribution of fluid, then, depends on vascular and other readjustments within the body.

It has been shown by Landis [9] that several types of hyperemia, such as those produced by heating the skin, increased the pressure within the capillaries of the skin. It is likely that the tendency of fluid to leave the blood stream that results from this increase of pressure has something to do with hastening evaporation from the surface.

When tannic acid was first introduced into the therapy of burns,[10] the belief was widely held that by coagulating the burned tissue it prevented the absorption of poisonous products and thus lessened the toxemia.[11] Underhill,[12] working with rabbits, offered evidence to contradict this hypothesis. He found that the absorption of certain dyes from the untreated burned surface was markedly retarded as compared to that from normal skin and that the extracts of burned skin and normal skin when injected into test animals produced like reactions. He explained the apparent toxemia on the grounds of altered circulatory relationships produced by concentration of the blood.[6]

Underhill and his associates [8b] and Blalock [8a] showed that after experimental burns in anesthetized animals edema fluid rapidly accumulates in adjacent tissues and that this fluid has essentially the same composition as blood plasma. In human beings this fluid forms large blebs, and some escapes, but in dogs, owing to the peculiar anatomy of the skin, it does not escape but is reabsorbed. Blalock found that the average total amount of fluid lost into inflamed tissues in eighteen dogs which had been burned over one-half the surface of the body was 57 per cent of the volume of the blood.

Treatment of burns with tannic acid reduces evaporation enormously, but the belief that the beneficial effects of tannic acid are due to the prevention of loss of water from the body is not supported by the data now available. Whatever water and protein are lost from circulation appear to remain largely within the body.

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8. (*a*) Blalock, A.: Experimental Shock: The Importance of the Local Loss of Fluid in the Production of the Low Blood Pressure After Burns, Arch. Surg. **22**:610 (April) 1931. (*b*) Underhill, F. P.; Fisk, M. E., and Kapsinow, R.: The Extent of Edema Fluid Formation Induced by a Superficial Burn, Am. J. Physiol. **95**:325, 1930.

9. Landis, E. M.: Micro-Injection Studies of Capillary Blood Pressure in Human Skin, Heart **15**:209, 1930.

10. Davidson, E. C.: Tannic Acid in the Treatment of Burns, Surg., Gynec. & Obst. **41**:202, 1925.

11. Martin, J. D., Jr.: Tannic Acid Treatment of Burns, South. M. J. **26**:321, 1930.

12. Underhill, F. P., and Kapsinow, R.: The Alleged Toxin of Burned Skin, J. Lab. & Clin. Med. **16**:823, 1931; footnote [6].

SUMMARY

Rates of evaporation were measured in thirteen cases of first degree and second degree burns, two cases of frost-bite, four cases of experimental burns produced by ultraviolet radiation and two cases in which tannic acid was applied to normal skin. In many instances corresponding cutaneous temperatures were measured.

It was found that the rates of evaporation from areas of first degree and second degree burns increased. Tannic acid formed (in second degree burns) a hard covering and prevented evaporation. In areas of first degree burns evaporation remained high. Evaporation from corresponding normal areas often increased somewhat for the first few days and then gradually returned to normal.

Cutaneous temperatures in burned as well as in normal areas were temporarily increased, corresponding in general to the rates of evaporation.

The failure of the rates of evaporation in areas of second degree burns to return completely to the normal level was correlated with the incomplete function of sweat glands, since no evidence of their activity could be demonstrated.

The changes in rate of evaporation and in cutaneous temperature in the cases of frost-bite and in the burns caused by ultraviolet radiation were essentially those of second degree and first degree burns, respectively. Tannic acid did not form a coagulum on areas of first degree burns, on irradiated skin or on normal skin.

When rates of evaporation were calculated for the whole body over a twenty-four hour period, it was ascertained that no significant changes in the weight of the body and no important losses in the total amount of fluid would be expected to result from evaporation alone.

CONSERVATIVE AND RADICAL MEASURES FOR TREATMENT OF ULCER OF THE LEG

II. A CRITICAL STUDY OF HEALING IN EXPERIMENTAL AND HUMAN WOUNDS UNDER ELASTIC ADHESIVE PLASTER

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NASHVILLE, TENN.

In the first report [1] under this general title conservative and radical measures for the treatment of ulcer were evaluated according to the results obtained from each. Indications were also suggested for their use. In the course of this study it was found that ulcers which were years old and which had been refractory to many other methods of treatment healed with striking rapidity when strapped with elastic adhesive plaster—a point previously noted by A. Dickson Wright, of England. Thus in this series three ulcers of an average size of 24.7 sq. cm., all of which had failed to heal under various remedies in an average time of nine and six-tenths years, actually healed under elastic adhesive plaster in an average time of fifty-one and three-tenths days. Results such as these stimulated interest in an attempt to throw light on the mode of action of elastic adhesive plaster, a dressing which although economical and easy to apply appeared to possess the remarkable properties already described for promoting the healing of wounds. The data which I have obtained from experiments on animals and human beings are detailed in the present report.[1a]

During the past two years I have quantitatively checked the healing of many ulcers of various kinds and sizes and have compared their rates of healing with the calculated or ideal curves of cicatrization given by Carrel and Hartman [2] and Du Noüy.[3]

From the Department of Surgery, Vanderbilt University.

1. Douglas, Beverly: Surg., Gynec. & Obst. **61**:458 (Oct.) 1935.

1a. In these experiments the bandages of three firms were employed, viz., the German preparation lomaplast, obtainable from G. Kuhlman, 62 W. 45th Street, New York; elastoplast, manufactured in this country and obtainable from the Duke Laboratories, Inc., Long Island City, N. Y., and an English preparation prepared from the formula of A. Dickson Wright and obtainable under the name of tensoplast from the Durex Products Corporation.

2. Carrel, A., and Hartman, A.: J. Exper. Med. **24**:429, 1916.

3. du Noüy, P. Lecomte: J. Exper. Med. **24**:451, 1916.

It will be recalled that by determining the surface area of sterile wounds by means of a planimeter these authors were able to derive simple and exact mathematical equations expressing the rate of healing. From these equations it is easy to predict the amount of healing which would take place in a given period of days for a sterile wound.

First it is necessary to obtain an index of cicatrization, i. This is calculated by equation (1):

$$(1)\quad \frac{\frac{S-S'}{S}}{t+\sqrt{T}} = i$$

Then the area of the wound after a given lapse of time may be calculated by a second equation, equation (2):

$$(2)\quad S'' = S'\,[1-i(t'+\sqrt{T+t'})]$$

Du Noüy explained the symbols as follows:

S represents the area of the wound at the beginning of the experiment.

S' represents the area of the wound *t* days later, at the time of the second observation. . .

t represents the time elasped between the first two observations, *S* and *S'*, in days.

T represents the age of the wound from the time of the first observation *S*. Therefore in the formula (1) $T = t$, and practically $t = t'$. . .

t' represents the time between the last observation *S'* and the time of the theoretical surface *S''* of the wound.

i is a constant coefficient characterizing each wound.

APPLICATION OF THE EQUATIONS TO THE PRESENT SERIES

At each visit excess secretions were blotted from the ulcers to be studied. Cellophane wound on cardboard spools and sterilized in an autoclave was unwound with sterile instruments and laid over the ulcer. The epithelial edge plainly visible through this was traced in a dotted line in ink with a fine pointed pen. The cellophane bearing the pattern was removed and stuck on a square of glass while the secretions were still moist on its under-surface. The hospital record sheet was then laid over the cellophane and the exact pattern traced on the record as silhouetted through the cellophane. Later this pattern was transferred by means of carbon paper to a sheet of standard bond paper of known weight and size. It was finally cut out of the bond paper and weighed. A simple division gave the exact area in square centimeters, and this in reality represented the actual area of the wound on the date on which the tracing was made. The details of this method for calculating the size of wounds are given in a previous article.[4]

4. Douglas, B.: Ann. Surg. 73:673, 1921.

COMPARISON OF THE RATE OF HEALING OR CICATRIZATION OF ULCERS UNDER ELASTIC ADHESIVE PLASTER WITH THE CALCULATED OR IDEAL RATE COMPUTED BY CARREL, HARTMAN AND DU NOÜY

Nine ulcers on the legs of seven patients have been specially studied. The ages of these patients as given on the charts varied from 13 to 69 years. Tracings were made at intervals of from seven to fourteen days by the gravimetric method as described, and actual areas of unhealed granulating surface were thus calculated. The ideal rates of healing were computed by equations 1 and 2 for comparison.[4a]

All the ulcers, even that of A.A., a 13 year old girl, were chronic and indolent. They had all been refractory to other methods of treatment. In spite of this seven of the nine actually healed more rapidly than sterile wounds of identical size would have healed according to the calculated ideal curves of healing (figs. 1 and 2).

The difference in healing time noted in favor of treatment with elastic adhesive plaster probably may be explained by the fact that in the care of the wounds studied by Carrel and his associates chlorine antiseptics, such as dilute solution of sodium hypochlorite, were commonly used to attain and maintain sterility; the recognized toxicity of these substances to living tissues, even though slight, was probably sufficient to retard the healing of the wounds and thus to influence the results of calculations.

Carrel and Dehelly [5] compared the action of petrolatum on one portion of a sterile wound on a man's arm with the action of dilute solution of sodium hypochlorite on another portion of the same wound. At the end of four days a tracing of the wound was made, and when this was compared with the preceding tracing, it was seen that the epithelial border had progressed a little more rapidly under the petrolatum than under the solution of sodium hypochlorite. At this time both the upper and the lower part of the wound were still aseptic. Carrel and Dehelly assumed, therefore, that the solution of sodium hypochlorite had slightly retarded the healing of the wound and was slightly toxic to the organism. Elastic adhesive plaster is coated with substances which seem absolutely nontoxic to tissue. It is probable, therefore, that formulas of healing derived from wounds treated with it will prove more accurate than those already derived by Carrel and his co-workers.

Certain it is that the composite geometric curve obtained by combining the graphs of healing in the cases of F. W. (right leg), P. H. (right leg), A. A., N. B., S. D., and H. P. would represent a more accurate picture of the healing of a sterile wound than would

4a. Mr. William W. Pyle checked the computations.

5. Carrel, A., and Dehelly, G.: Treatment of Infected Wounds, New York, Paul B. Hoeber, Inc., 1919, p. 37.

the composite graph of Carrel and Hartman's curves. It is noted that in the latter graph the curve is not as steep as, and reaches the abscissa at a later date than, in the graph for healing of wounds in treatment of which no antiseptic was used (figs. 1 and 2).

It is believed that the facts that healing of these old ulcers of the leg is so rapid under elastic adhesive plaster, a chemically inactive dressing the properties of which are admittedly almost entirely physical, and that dilute solution of sodium hypochlorite is admittedly slightly toxic to tissues both indicate that progress in the development of therapeutic measures to promote or aid healing of wounds will be made in the future along physical rather than along chemical lines.

One point is believed to be of great importance, namely, that, as definitely shown by Carrel and Hartman,[2] contraction is the more important factor in healing of wounds and that epidermization completes the work of contraction in healing. It is likely that with elastic adhesive plaster the constant pull inward (at least in one dimension or direction) tends to promote contraction in tissues which have been prevented from exercising this important function in healing by such factors as edema and excessive deposits of scar tissue.

In any event it seems astounding that these large, chronic ulcers should have healed more rapidly than if they had been fresh sterile wounds.

Two ulcers did not heal as calculated ideal curves indicated that they should have healed. One of these had been present in a man of 39 for ten years and was surrounded by a dense and deep scar. Du Noüy's formula indicated that it should have been healed on the twenty-third day. A tracing on the twenty-seventh day showed that it measured 4 sq. cm., and on the fiftieth day it still measured 0.33 sq. cm.; after that I did not see the patient (fig. 1 *B*). Meanwhile a much larger ulcer on the other leg healed more rapidly than the ideal curve indicated that it should, showing that some local factor which was not discovered caused the first ulcer to heal so slowly (fig. 1 *C*).

The other patient, a Negro aged 58, had a nonspecific elephantiasis from an old thrombophlebitis and two ulcers which were of twenty years' duration. One ulcer healed under elastic plaster more rapidly than the curve of ideal healing indicated that it should (fig. 2 *C*). The other ulcer rapidly diminished in size and measured only 1.5 cm. in twenty-five days, but a small deep area remained unhealed under this treatment until fourteen months later, when strapping with a pad over the plaster was successful in accomplishing complete cicatrization (fig. 2 *E*). I am certain now that the ulcer would have healed more promptly if the application of the pad had been resorted to sooner.

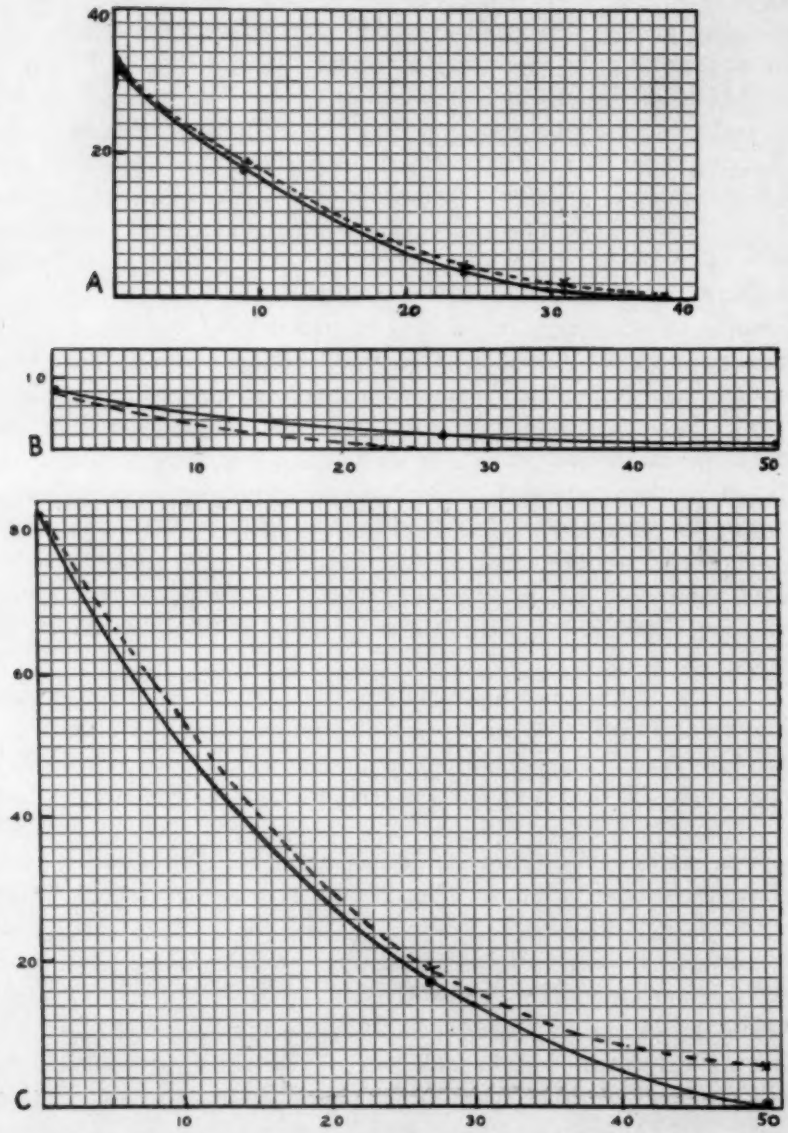

Fig. 1.—Curves showing healing of ulcers. *A*, curve for ulcer in H. P. ($i = 0.038$); *B*, curve for ulcer on the left leg of F. W. ($i = 0.05$); *C*, curve for ulcer on the right leg of F. W. ($i = 0.025$). The solid line represents the actual curves of healing or cicatrization; the dotted lines represent the ideal curves of healing as computed by Carrel, Hartman and du Noüy. The vertical units represent areas in square centimeters; the horizontal units represent days.

MODE OF ACTION

The general action of elastic adhesive plaster in compressing dilated veins and thus improving the nutrition of the tissues is like that of the elastic stocking and other similar devices. Owing, however, to its

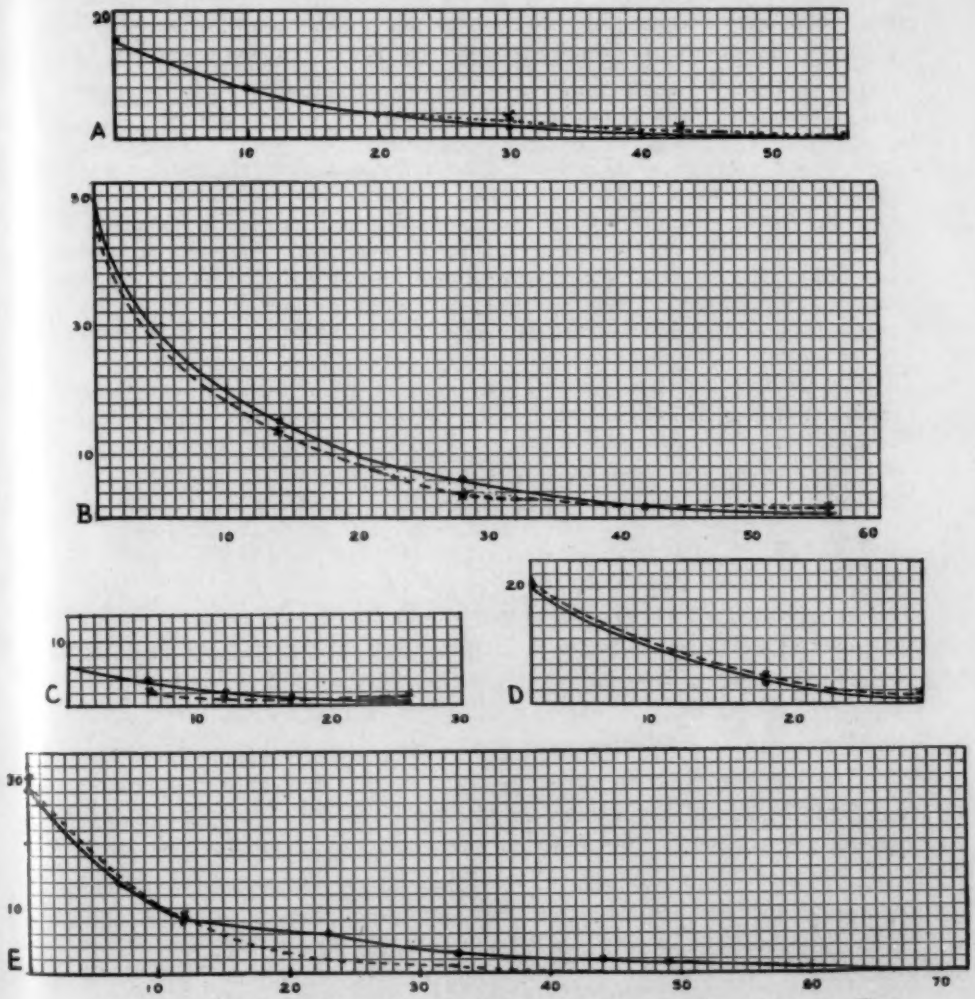

Fig. 2.—Curves showing healing of ulcers. *A*, curve for ulcer in S. D. (i = 0.032); *B*, curve for ulcer in N. B. (i = 0.039); *C*, curve for ulcer on the right leg of P. H. (i = 0.025); *D*, curve for ulcer in A. A. (i = 0.33); *E*, curve for ulcer on the left leg of P. H. (i = 0.19). The vertical units represent areas in square centimeters; the horizontal units represent days.

adhesive coating it holds securely to the leg and maintains a more even and constant pressure on varicosities until the latter can be treated by curative measures.

Several aspects of the local action of elastic adhesive plaster on the wound proper appear to be responsible for the remarkable rapidity with which healing is accomplished.

In the first place, it provides moist healing conditions without permitting the formation of a dead space. Plasma and pus when excessive may filter out of the minute spaces between the threads of waterproofed cloth. It is therefore semipermeable. In 1913 Halsted[6] pointed out the advantages of maintaining the moisture of the edges of the wound during healing in the following words:

> A moist scab, so-called, is better than a dry one. The surgeon has many opportunities to convince himself of this. When he has occasion to extract a toe-nail let him cover one-third of the raw surface with protective [gutta-percha tissue] and allow the remainder to heal under the dry scab. The patient may have pain in the digit soon after the dry scab is well formed, and the surgeon will perhaps observe that the soft parts about the nail-bed on the side of the dry scab are inflamed. On covering the entire surface of the granulating wound with gutta-percha tissue the pain will be relieved and the inflammation subside.
>
> The dry scab imprisons secretion which in turn gives rise to tension, and the tension impairs the vitality of the tissues under and at the border of the dry scab to an extent sufficient to place them at a disadvantage with the micro-organisms and to compel absorption of the toxins.

Elastic adhesive plaster does not imprison secretions to any appreciable extent, for the exudate comes out through the plaster as well as around its edges. It does, however, provide moist healing conditions—"a moist scab, so-called"—the advantages of which Halsted so well emphasizes in this description. That the irritation and pain so commonly experienced with a dry dressing are promptly relieved by elastic adhesive plaster is attested by the repeated statements of patients who have had it applied to their ulcers.

Another action of a dressing such as this which provides moist conditions was not specifically mentioned by Halsted, namely, that by protecting the surface from recontamination or reinfection by outside organisms it permits the blood to build up at the wound antibodies definitely specific for the particular organisms present at first. This is an aid to rapid sterilization and thus to rapid healing.

One disadvantage of a dressing which fails to maintain moist conditions on the surface of the wound is that drying destroys the leukocytes and must also destroy antibodies. This was emphasized by A. Dickson Wright in a recent article.[7] Thus the forces of resistance of the body to micro-organisms are broken down by a drying of secretions.

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6. Halsted, W. S.: Ligature and Suture Material, J. A. M. A. **60**:1119 (April 12) 1913.

7. Wright, A. Dickson: Brit. M. J. **2**:561 (Sept. 26) 1931.

Elastic adhesive plaster obviates this by keeping the wound moist at all times without permitting the formation of an appreciable dead space. Its value from this standpoint is obvious.

Bacteriologic studies bearing on this point are not yet complete, but I report the following case, which was studied fully by Dr. Robert Schrek and me.

E. W., a college girl aged 24, scratched her legs while walking through briars on about Aug. 3, 1933. Ulcers developed at the sites of the scratches, and on admission to the hospital on August 9 there were sixteen ulcers on her legs. The two largest were chosen for special study. By reference to the photographs the method of procedure and the results may easily be followed. For purposes of clarity hereafter the ulcer chosen for observation on the right leg will be designated as *a* and the one at a corresponding level on the left leg as *b*.

Fig. 3.—Bilateral ulcers on the legs of E. W. At left, condition at start of treatment (September 11); at right, ulcer, *a*, on the right leg, which was treated with a solution of boric acid, is still open; ulcer, *b*, on the left leg, which was treated with elastic adhesive plaster, is healed. See also table 2.

From figure 3 it is readily seen that on September 11 *b* was almost twice as large as *a*. The subsequent treatment and results are shown in table 1.

Thus, *b*, a larger ulcer than *a*, when treated by elastic adhesive plaster became sterile in nine days (or twelve days earlier than *a*) and healed seventeen days sooner than *a*, which was treated by gauze packs moistened with warm saturated solution of boric acid.

Another property of elastic adhesive plaster which I consider valuable and which I am therefore studying rather intensively is its persistent elastic pressure on the wound, an action which for want of a better term I call a "gloving action." I have been forced recently to conclude that this plays a major rôle in the actual speeding up of the

healing process. How does it do it? The essential rôle that moderate pressure plays in promoting "takes" in whole thickness skin grafts by insuring contact and lessening inflammation is well known. I am certain that a similar rôle is played by pressure in the healing of ulcers without grafts. First, if proud flesh, or exuberant granulation tissue, is present its further formation is checked and that already formed is pressed back by the constant elastic pressure. This rapidly produces a granulating surface level with the epithelium and favors the latter's rapid advance over it. Conversely, the epithelial edge surrounding a depressed wound will be gradually pressed down to the lower level of the granulating surface.

By accident in August 1932 I dressed a patient on whose leg an elastic adhesive bandage had been improperly applied. The turns of the bandage had been made to overlap each other too little. The leg was

TABLE 1.—*Comparison of the Effect of Saturated Solution of Boric Acid with That of Elastic Adhesive Plaster on Bacteria and Healing of Ulcers of E. W.*

	Ulcer *a* *		Ulcer *b* †	
Date	Number of Colonies on Culture	Size	Size	Culture
9/11	Innumerable	Small	Large	Infinity
9/16	Innumerable	Small	Large	5 colonies
9/20	Innumerable	.....	Small	Sterile
10/ 2	Sterile	Small	Small	
10/ 4	.....	Small	Healed	
10/21	.....	Healed		

* Ulcer *a* was treated with a solution of boric acid.
† Ulcer *b* was treated with elastic adhesive plaster.

therefore found to have been pressed too hard in certain places, while little if any pressure had been exerted in others. Multiple ulceration was present. When the bandage was removed an interesting and instructive phenomenon was seen. Wherever the turns of the bandage had pressed firmly against the tissues the ulcers were healed. On the contrary, wherever the turns had been too loose the granulations were exuberant, and epithelium had not grown over. In some places these two conditions were seen side by side in the two halves of a single ulcer. A photograph of the wound shows this clearly.[8] The rôle of the elastic adhesive bandage in producing elastic pressure to hold down granulations was thus excellently demonstrated.

8. Since this report was submitted I have read with interest an article by E. D. Twyman published in the Journal of the Missouri State Medical Association (**19**:1 [June] 1922), entitled "Epithelial Proliferation, Differential Pressure as an Aid in the Growth of Epithelium on a Denuded Surface." Twyman expressed the belief that epithelium proliferates readily when it is subjected to a degree of pressure which restrains the growth of granulations. The report contains a photograph of a wound showing "a bar of epithelium grown across from side to side

CONTRACTION OF TISSUES

The function that contraction plays in healing, as emphasized by Carrel and Hartman, has already been mentioned. It is obvious that the concentric pull of elastic adhesive tape must greatly favor this and must therefore be important in promoting healing of wounds.

In eight cases they studied the parts that contraction and epidermization each play in the healing process. For the purpose of discussion two of their charts are reproduced.[2]

The first of these, figure 4, represents the graphic curve of healing of a wound of the upper clavicular region. The lower curve represents the total surface area of the unhealed or granulating portion of the

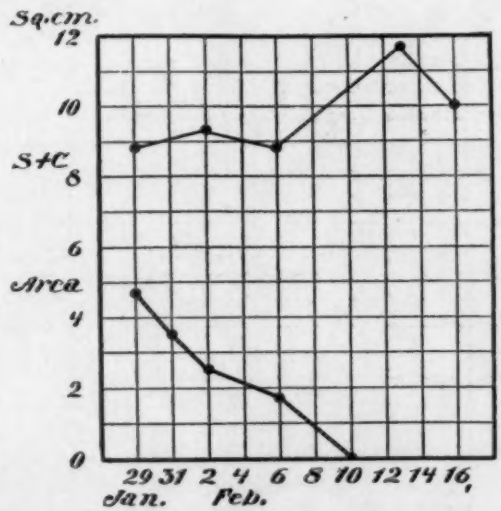

Fig. 4.—Graphic curves of healing of a wound of the upper clavicular region. The graph is figure 19 of the article by Carrel and Hartman.[2]

wound. The upper curve represents this plus the area of the scar already formed. It was obtained by tracing the outer border of the scar or cicatrix on cellophane and determining the area included by the use of a planimeter. It is seen, as Carrel and Hartman pointed out, that the upper curve remains horizontal for a while and afterward inclines upward. Cicatrization or healing in this case was therefore due entirely to epidermization and not to contraction. During the

by strapping across it" with ordinary nonelastic adhesive plaster. His observations were well made, and I agree with him as to the value of ordinary adhesive plaster. This work should go far toward substantiating the view expressed first by Morison and then by Carl Beck, who advocated treatment of superficial wounds by means of strapping with adhesive plaster. It is in direct line with my observations on the healing of wounds under elastic adhesive plaster.

latter days of healing it is seen that the area of the scar actually enlarged, which means that since contraction was lacking, epidermization became more rapid and compensated for the dilatation of the cicatrix.

The second chart, or figure 5, represents the curve of healing of a shell wound of the external part of the leg in a patient 39 years of age. It is seen to be quite different from that in figure 4. Thus, between January 3 and 15 contraction alone was largely responsible for healing. Between January 15 and 19, since the upper curve remains horizontal, the downward deflection of the lower indicates that epidermization was responsible for healing. After this both processes took part in the healing of the wound, though epidermization assumed the major rôle.

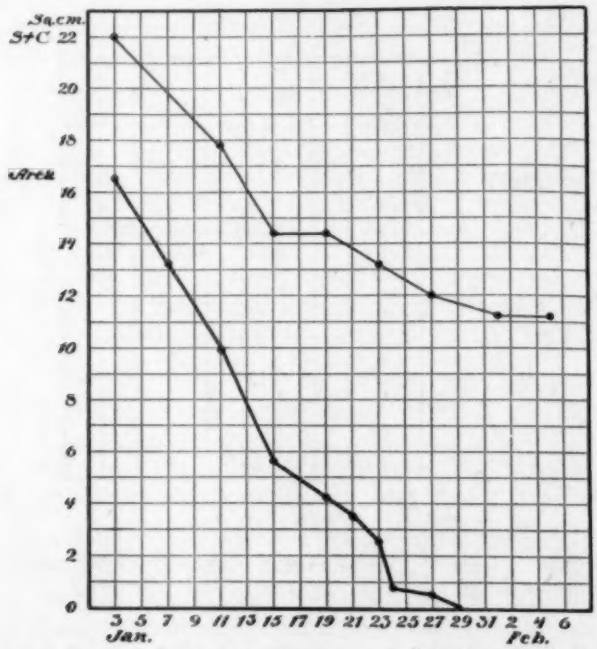

Fig. 5.—Curve of healing of a shell wound of the external part of the leg in a patient 39 years of age. (The graph is figure 21 of the article by Carrel and Hartman.[2])

From these curves, then, it can be seen that two processes, contraction and epidermization, contribute to healing of wounds in varying degrees, according to the particular wound. Carrel and Hartman stated that contraction, as a process, is dependent on contraction of granulation tissue and on the opposite action of the tissues surrounding the wound. According to them, the resistance of the skin surrounding the wound is slight in narrow wounds, and in larger defects the wound contracts until the elasticity of the surrounding skin prevents a greater reduction of its size.

These facts may easily be verified by considering the pulling apart or retraction of edges of skin which occurs when a circle of skin is excised from a dog's abdomen. The outline of the pattern (fig. 6, experiment A) along which the circular incision was made measured 5.2 cm. in its transverse diameter, but after the central skin was excised it measured 6.3 cm. Every surgeon is well acquainted with the gaping of the edges of a wound which occurs when a simple line incision is made and knows how large an area from which a full thickness graft is cut may become by retraction of edges. Traction by adhesive tape is often used in the surgical treatment of wounds resulting from accidents in place of sutures to decrease the size of skin defects. It is probable that elastic adhesive plaster may accomplish similar results more gradually in older wounds by slowly overcoming the outward pull of the surrounding skin.

In certain old ulcers a share of the failure to heal or tardiness in healing which occurs at times may be due to the fact that granulations are unhealthy and fail to contract. It is wholly logical that elastic adhesive plaster may supply the force to contract and heal which is lacking in such cases.

Knowing these facts, I decided to investigate the exact effect of the constant mechanical tension of elastic adhesive plaster in one dimension toward the center of the wound on the process of contraction.

EXPERIMENTAL EVIDENCE THAT ELASTIC ADHESIVE STRAPPING FAVORS HEALING THROUGH CONTRACTION

EXPERIMENT A.—Three oval outlines were marked off on the abdomen of a dog after it had been shaved. With the animal under ether anesthesia the skin was excised between the two outer outlines and inside the inner one, thus leaving a circular band or "doughnut" of skin isolated on a raw surface which was to serve for a different study. Tracings on cellophane of the epithelial edge of the outermost circle were made regularly every two days as the edge grew inward toward the center. Tracings on the same cellophane were made of the outline of the circle of skin as the edges of the latter grew inward and outward, respectively. At each dressing the wound was covered with dry sterile gauze strapped on with adhesive plaster.

The progress of healing is shown by the reduced drawings (fig. 6, experiment A). In the last tracing the heavier outer ring represents the outline of the wound at the start of the experiment. The inner dotted lines represent the degree of advancement of the healing edges twelve days later.

From these drawings it is seen that the shape of the wound, an oval with the greatest dimension vertically placed, remained much the same during healing when an ordinary type of dressings was used. Measurements showing this to be true are given in table 2.

As the wound healed, therefore, the vertical diameters were reduced almost exactly proportionately to the horizontal diameters, the ratio 4.9/4.2 being approximately equal to 2.7/2.5.

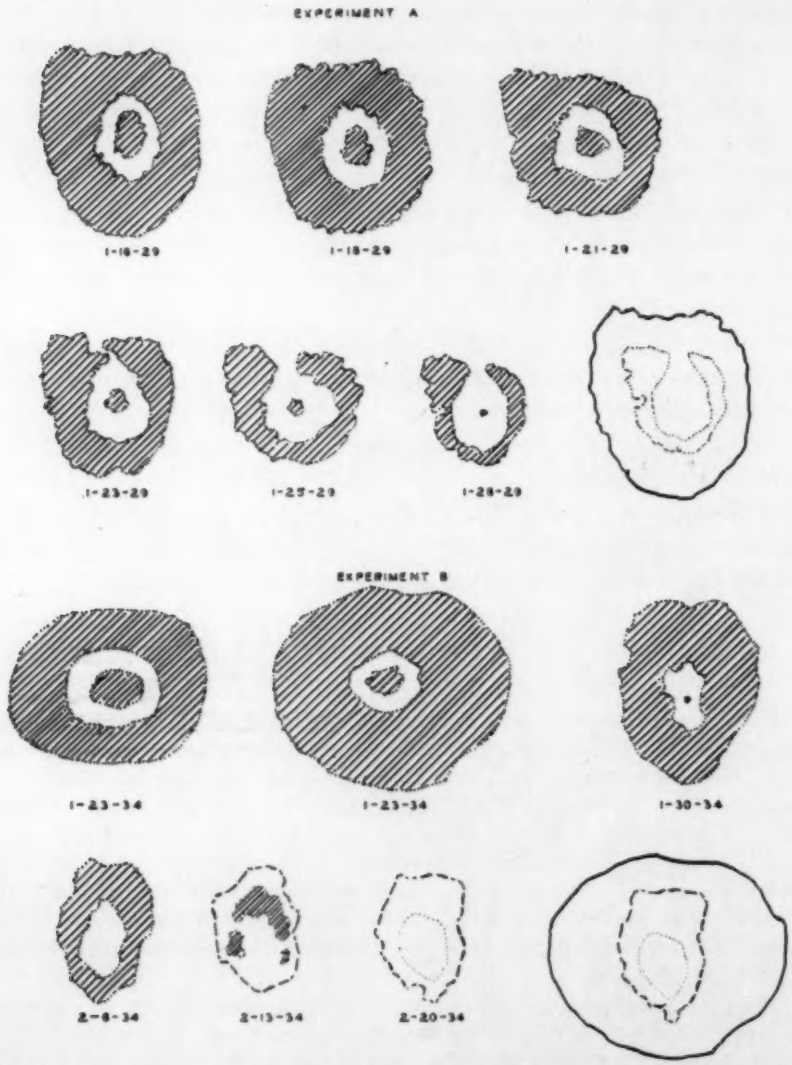

Fig. 6.—Diagrams showing healing of a ring of skin on a granulating surface (experiment A) and healing of a ring of skin under elastic adhesive plaster (experiment B).

EXPERIMENT B.—On a dog anesthetized with ether a wound similar to that of the dog in experiment A was made by excision of skin from the abdomen. In this experiment, however, the outer outline was made larger and the greatest

diameter of the oval was placed horizontally instead of vertically. Similar tracings were made throughout, but in addition the outline of the cicatrix was traced so as to determine how much contraction of the scar, if any, occurred. Instead of the ordinary dry gauze dressings used in experiment A, elastic adhesive plaster was stretched firmly in a horizontal direction across the wound and was replaced at each tracing. Figure 6 (experiment B) shows the progressive changes in outline during healing. The heavier outer ring in the last tracing represents the outline of the wound at the start. The inner heavier dotted outline represents the advancement of the edge after fourteen days of healing. The innermost circle shows the final shape and size of the cicatrix.

It may be noted that the shape of the wound was changed by elastic adhesive strapping from a horizontal oval to a vertical oval.

Results.—The results as given in tables 3 and 4 are self-explanatory.

Thus it is seen that the shape of the wound was changed during healing in such a manner that the horizontal diameter, which was originally greater, became smaller than the vertical. The original fraction representing the relation of the vertical to the horizontal dimension changed from 5.3/6.2 to 4.6/3.6.

TABLE 2 (*Experiment A*).—*Linear Measurement of Greatest Diameters of the Wound in Centimeters*

Date	1/16	1/18	1/21	1/23	1/25	1/28
Vertical measurement	4.9	4.5	3.6	3.7	3.1	2.7
Horizontal measurement	4.2	4.2	3.9	3.4	3.4	2.5

When one turns attention to the cicatrix or scar, it is found that a similar condition exists. The original fraction representing the relation of the vertical to the horizontal dimension changed from 5.3/6.2 to 3.3/2.3. The greatest decrease in the size of both the granulating surface and the scar in experiment B is seen to occur in the horizontal dimension, in which the inward pull of the elastic adhesive bandage was exerted. Since there was no corresponding change in either diameter of the wound in experiment A, which was treated without elastic adhesive bandage, it is concluded that the latter is responsible for the diminution in size or the contraction produced. Also, since it was found that the healing was much more rapid in the wound treated with elastic adhesive bandage, it was concluded that the contraction which was shown to be present must play a favorable rôle in healing.

Evidence from Human Wounds.—I have made similar studies based on human wounds of the rôle of contraction or shrinkage of wounds due to the pull inward of elastic adhesive strapping which is applied spirally about the lower extremity. The patients in each case had one or more ulcers of the leg. The elastic adhesive bandages were applied

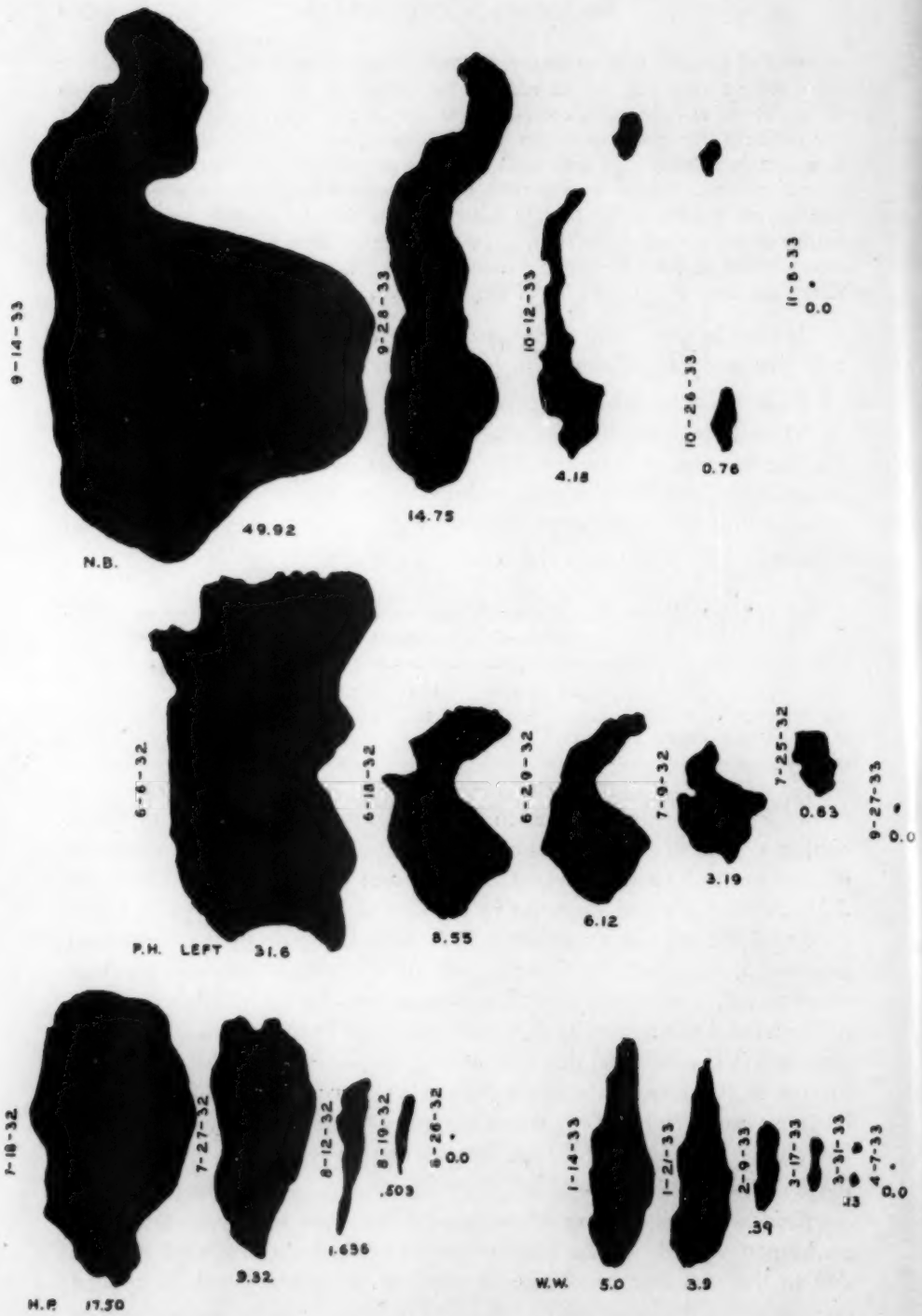

Fig. 7.—Cut-outs showing progressive healing of human ulcers. The horizontal numbers indicate areas of wounds in square centimeters; the vertical numbers show the dates.

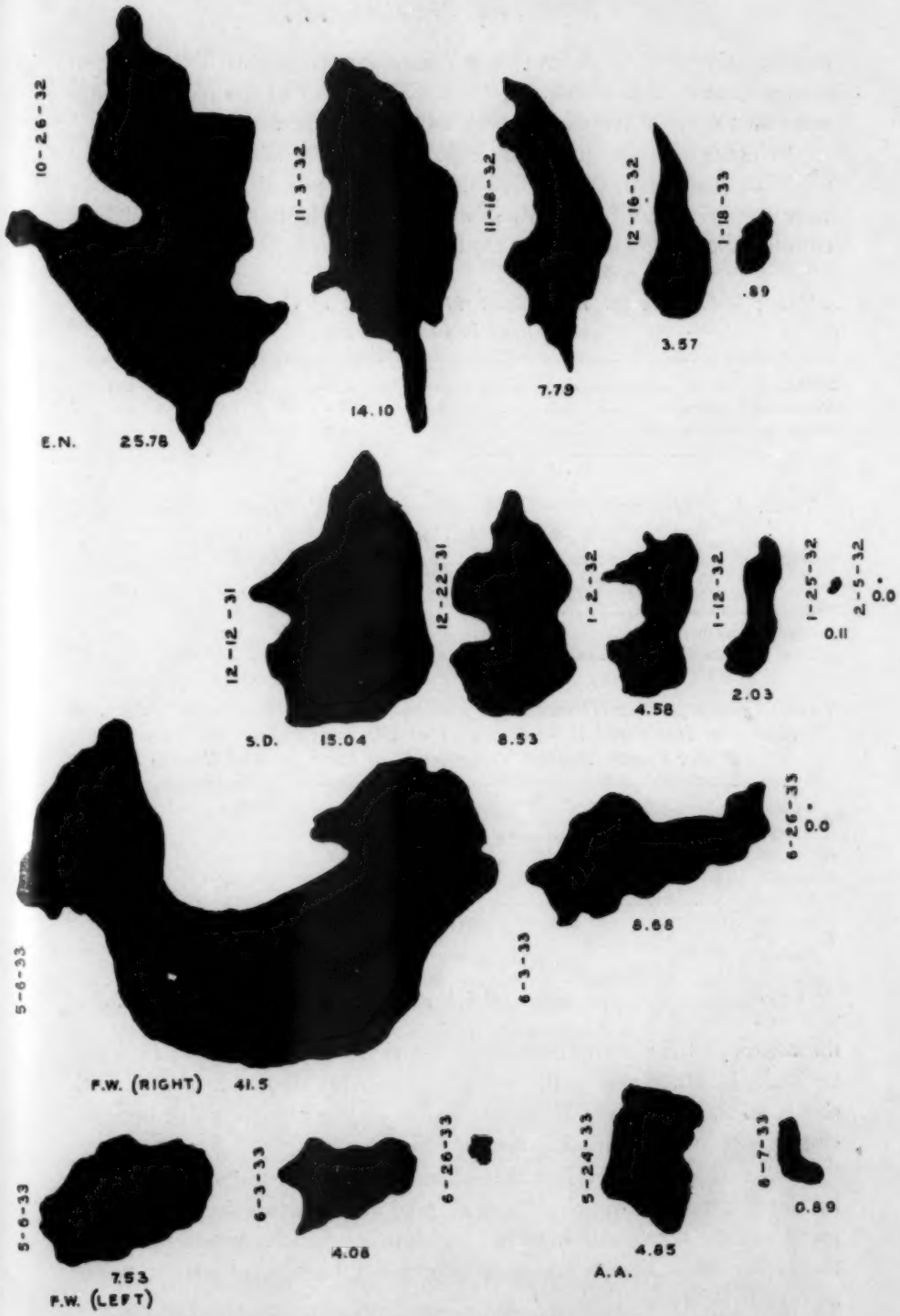

Fig. 8.—Cut-outs showing progressive healing of human ulcers. The horizontal numbers indicate areas of wounds in square centimeters; the vertical numbers show the dates.

transversely so as to produce a pressure inward toward the center of the leg of about 2 to 3 pounds (0.9 to 1.3 Kg.) per square inch. Tracings were then made at regular intervals as the ulcers healed.

In order that the progressive decrease in the areas of the wound might be visualized, cut-outs of their outlines placed over black paper were employed. A few of these are shown in figures 7 and 8. These cut-outs show quite a uniform tendency for the transverse or horizontal

TABLE 3 (*Experiment B*).—*Linear Measurement of Greatest Diameters of Wound in Centimeters*

Date	1/23	1/30	2/6
Vertical measurement	5.3	4.8	4.6
Horizontal measurement	6.2	3.9	3.6

TABLE 4 (*Experiment B*).—*Linear Measurement of Greatest Diameter of Cicatrix in Centimeters*

Date	1/23	2/13	3/20
Vertical measurement	5.3	3.3	3.3
Horizontal measurement	6.2	2.2	2.3

TABLE 5.—*Comparative Decrease in Maximum Vertical Dimension (V) and Maximum Horizontal Dimension (H) of Ulcers in Centimeters (Elastic Adhesive Plaster Applied Transversely to Long Axes of Ulcers)*

Case	V^1	V^2	H^1	H^2
H. P. (25)*	6.4	3.5	3.7	0.7
W. W. (26)	5.2	2.1	1.4	0.4
S. D. (30)	5.8	2.9	3.2	0.9
P. H. (33)	3.2	1.7	1.7	0.7
N. B. (14)	12.4	9.6	7.3	2.5
Totals	33.0	19.8	17.3	5.2

* The figures in parentheses denote intervals in days between measurements in each case.

dimension of the ulcer to decrease proportionately more rapidly than the vertical. In other words, the wounds in general assume a long narrow shape, so that the edges lying lengthwise of the extremity are brought close together before healing takes place.

From table 5 it is evident that under the effect of transverse traction by elastic adhesive strapping the widths of all the ulcers decreased much more rapidly than their heights. By figuring out the proportions, the transverse dimension is found to decrease 1.9 times, or almost twice, as rapidly as the vertical.

In the case of N. B. linear measurements of the outline of the scar or cicatrix gave the values shown in table 6. These values indicate that healing in this case proceeded by means of both contraction and epidermization but that the former must have paved the way for the latter by helping to render the wound a long narrow one. Since the contraction is seen to have occurred almost totally in one dimension, viz., the horizontal, it seems certain that the transverse concentric pull inward of the elastic adhesive plaster must here, as in all the cases previously referred to, have exerted a favorable influence on the factor of contraction.

Since the elastic adhesive plaster used in these studies exerts a force in only one direction, the question logically arises: What would

TABLE 6.—*Linear Measurements of Greatest Diameter of Cicatrix in Centimeters (N. B.)*

Date	9/14	9/28	10/12	12/13
Horizontal measurement	7.3	5.4	4.6	4.3
Vertical measurement	12.4	11.8	12.2	12.0

TABLE 7.—*Comparative Decreases in Maximum Vertical Dimension (V) and Maximum Horizontal Dimension (H) of Ulcers in Centimeters (Elastic Adhesive Plaster Applied Lengthwise to Long Axes of Ulcers)*

Case	V^1	V^2	V^3	H^1	H^2	H^3
F. W.	3.9	2.5	0 (healed)	10.5	5.4	0
Q. W.	2.3	1.7	0.55	3.9	2.9	0.5
B. A.	1.1	0.5	0.35	1.7	1.1	0.9
Totals	7.3	4.7	0.9	16.1	9.4	1.4

be the effect of applying the plaster so that its pull is inward in the direction of the long dimension or axis of a wound instead of in the short axis? In other words, what would be the effect of applying the plaster so that its pull centrally would be exerted lengthwise of a long narrow wound? Unfortunately most patients available for study had ulcers of the legs, and these were narrower in the long axis of the leg than they were in the short transverse axis, or the direction in which the elastic adhesive plaster was stretched to encircle the leg.

The opposite was true in three cases, however. In these the ulcers were long in the transverse axis of the leg and comparatively narrow. The elastic pull of the adhesive plaster was exerted inward along their long axes.

Table 7 shows that in spite of the pull of the elastic adhesive plaster being inward along the lengths of the ulcers the latter remain relatively

long and narrow during healing, or, in other words, in general they retain their original shapes. These shapes, incidentally, have been shown to be the ones best adapted for the attainment of rapid healing.

It is to be noted in this connection that while the lengths of the narrow wounds are not sufficiently decreased to alter completely the shapes of the wounds, nevertheless they show decreases which are marked when considered relative to the changes in wounds not treated with elastic plaster. If healing had not supervened too early, their shapes would probably have been altered. The case of Q. W. corroborates this, since just before healing the wound assumed an almost circular shape, with a vertical diameter of 0.55 cm. and a horizontal diameter of 0.5 cm.

The data on experimental wounds and on wounds in human beings which we have presented show that elastic adhesive plaster favors contraction in one axis of the wound and through this action greatly favors the healing process.

One more question seems pertinent to the discussion. Since it is known that a surface wound such as an ulcer has two dimensions and elastic adhesive plaster can contract in only one of these, why would not more rapid healing be obtained by using a material uniformly elastic in all directions, such as finely perforated dental rubber-dam impregnated with the same kind of plaster? Such a plaster would shrink the wound inward in all directions. In answer, I may say that I have already used strips of elastic adhesive strapping vertically as well as longitudinally simultaneously over wounds by stretching a strip up the side of the leg and covering it with the usual spiral turns. The effect seems to be better than that produced by using the spiral turns alone. It is readily seen, however, that since the cloth is not elastic in the direction of its width this nonelasticity must necessarily impede the pull inward in this direction of the overlying cloth.[9]

SUMMARY AND CONCLUSIONS

In a previous report, prompt healing under elastic adhesive plaster was reported in 96.7 per cent of chronic ulcers, most of which had been refractory to other nonoperative forms of treatment.

A closer study of a typical group of ulcers by quantitative methods shows that: (*a*) The healing rate of most chronic ulcers treated by elastic adhesive strapping is more rapid than the ideal rate of healing for recent wounds kept sterile by dilute solution of sodium hypochlorite

9. Since this article was submitted I have constructed a semipermeable elastic adhesive cloth which is uniformly elastic in all directions. This has been named equiplast. The effect which it has already produced on experimental and human wounds will be made the subject of a further report.

as computed by Carrel, Hartman and du Noüy; (*b*) du Noüy's ideal algebraic equations for the rate of healing or cicatrization of sterile wounds must be corrected for a constant error when physical rather than chemical agents (antiseptics) are employed.

New geometric curves are derived which it is claimed express more accurately the ideal rate of wound healing than those formerly given by Carrel and Hartman.

The several actions through which elastic adhesive plaster exerts a favorable influence on wound healing are critically analyzed.

SHOCK: THE MECHANISM OF DEATH FOLLOWING INTESTINAL OBSTRUCTION

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The mechanism by which death results from intestinal obstruction remains a much debated question. The literature on this subject is voluminous, and extensive experimentation has led to dissension rather than to agreement. The reports are characterized by general similarity in the essential results of experimentation and by diversity of interpretation. No attempt will be made here to analyze these reports. The analysis by Cooper [1] considered 161 articles, and reflected the divergent views of the authors. He reviewed the evidence and interpretations and closed his summary with the unanswered question: "What is the cause of death in high intestinal obstruction?"

In studies on shock of various origin in man and on experimental shock, we are struck by the similarity between that condition and the phenomena resulting from intestinal obstruction. When shock is induced in dogs without trauma and without the complicating factor of deep narcosis, a characteristic group of symptoms is produced. The animal becomes inactive and listless and refuses food. There are muscular tremors, vomiting, salivation and, in the later stages, the passage of fluid stools, which frequently contain blood and mucus. The pulse becomes rapid, the blood pressure falls, the temperature becomes subnormal or sometimes increased and the respirations are slow and deep. The blood becomes markedly concentrated, the nonprotein nitrogen content increases and the coagulability decreases. The urine becomes scanty and concentrated. Frequently it contains blood and albumin. Lethargy or coma and evidence of circulatory failure precede death.

This syndrome as described may be produced by introducing fresh finely chopped dog muscle into the peritoneal cavity, by the intravenous injection of peptone, of watery extracts of various tissues, of histamine, mercury bichloride or various other drugs or of sterile suspensions of bacteria or bacterial products and by other means.

This work was aided by the Martin Research Fund.

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1. Cooper, H. S. F.: Cause of Death in High Obstruction, Arch. Surg. **17**:918 (Dec.) 1928.

In articles dealing with intestinal obstruction a few of the authors have commented on the shocklike manifestations, but the majority of the reports have contained no suggestion that the shock syndrome has been recognized. There is general agreement that in dogs with intestinal obstruction at a high level there are vomiting, tremors and weakness, the pulse becomes rapid and weak, and the blood pressure falls. The blood becomes concentrated, its nonprotein nitrogen content increases and the chloride content decreases. The urine is scanty and may contain albumin or blood. Coma or lethargy precedes death. There is agreement that the same features follow the intravenous injection of various preparations of intestinal contents from a closed or strangulated loop. Stone and his associates [2] attributed this to alteration of digestive secretions and perhaps to absorption of products of protein cleavage from the closed loop. Gerard [3] attributed the intoxication to the liberation of histamine and other products of protein cleavage from the wall of the obstructed bowel. He showed that both histamine and substances from obstructed bowels produce a fall in blood pressure and temperature, rapid respiration, vomiting, bloody diarrhea, decreased coagulation of the blood and hemoconcentration. Marked engorgement of the gastro-intestinal mucosa was recorded as a postmortem feature following such injections. Some workers produced these manifestations by intravenous injections of bacteria or their products. Numerous experiments of these types are on record. Apparently none of the authors were aware that each of these procedures will produce shock and that the manifestations described are characteristic of the shock syndrome. Detailed descriptions of the postmortem observations, with particular reference to visceral circulatory changes, would be most enlightening in such experiments. Unfortunately, these were not included in the published data.

CHANGES CHARACTERISTIC OF SHOCK

Most of the studies on intestinal obstruction were published before it was recognized that shock is accompanied by characteristic changes demonstrable by postmortem examination. It seems remarkable that these changes have so long escaped the attention of those interested in such conditions. Heubner (1907) [4] produced fatal shock by intravenous injection of gold chloride and gave the earliest description we have found of the visible changes characteristic of shock. The finest venules, especially in the abdominal viscera, were heavily congested.

2. Stone, H. B.; Bernheim, B. M., and Whipple, G. H.: Bull. Johns Hopkins Hosp. **23**:159, 1912; Ann. Surg. **59**:715, 1914.

3. Gerard, R. W.: Lethal Agent in Acute Intestinal Obstruction, J. A. M. A. **79**:1581 (Nov. 4) 1922.

4. Heubner, W.: Arch. f. exper. Path. u. Pharmakol. **56**:370, 1907.

The peritoneal surfaces of the bowels were rose-red or bluish red. The mucosae were dark red and contained numerous hemorrhagic flecks. Similar minute hemorrhages were present in the surfaces of the liver, spleen and kidneys. Blood-tinged fluid was present in the serous cavities. The lungs were deep red and contained numerous ecchymoses in their substance and also in the pleura. Blood flowed freely from the cut surfaces of the organs. The outstanding microscopic features were distention and engorgement of the minute vessels in all the tissues. There were numerous capillary hemorrhages in the substance of the organs, especially in the lungs, liver and kidneys. Heubner concluded that circulatory failure resulted from the widespread dilatation of the capillaries and venules. The animals had bled to death into their own capillaries, as he expressed it.

Dale and Laidlaw [5] noted similar circulatory changes in shock following injections of histamine. An increase in the concentration of hemoglobin and red cells of 40 per cent was an associated feature.

Observations on congestion, edema, capillary hemorrhages and hemoconcentration (increase in the concentration of hemoglobin and red cells) were recorded by many authors (Hooker,[6] Gasser and his associates,[7] Keith,[8] Bayliss,[9] Cannon and his co-workers,[10] Whipple and his co-workers [11]) in studies on traumatic shock. In the records of postmortem examinations after shock from battle wounds, marked congestion and edema of the viscera, effusions of fluid into serous cavities and capillary hemorrhages in mucous and serous surfaces were noted

5. Dale, H. H., and Laidlaw, P. P.: J. Physiol. **52**:110 and 355, 1919.

6. Hooker, D. R.: Am. J. Physiol. **54**:30, 1920.

7. Gasser, H. S.; Erlanger, J., and Meek, W. J.: Am. J. Physiol. **49**:90 and 151, 1919; **50**:31, 1919.

8. Keith, N. M.: Blood Volume in Wound Shock, in Reports of the Special Investigation Committee on Surgical Shock and Allied Conditions: VIII. Traumatic Toxaemia as a Factor in Shock, Medical Research Committee, Special Report Series, no. 26, London, His Majesty's Stationery Office, 1919, p. 36.

9. Bayliss, W. M.: Observations on the Results of Muscle Injury and Their Treatment, in Reports of the Special Investigation Committee on Surgical Shock and Allied Conditions: VIII. Traumatic Toxaemia as a Factor in Shock, Medical Research Committee, Special Report Series, no. 26, London, His Majesty's Stationery Office, 1919, p. 23.

10. Cannon, W. B.: Some Characteristics of Shock Induced by Tissue Injury, in Reports of the Special Investigation Committee on Surgical Shock and Allied Conditions: VIII. Traumatic Toxaemia as a Factor in Shock, Medical Research Committee, Special Report Series, no. 26, London, His Majesty's Stationery Office, 1919, p. 27; Traumatic Shock, New York, D. Appleton & Company, 1923. Cannon, W. B.; Fraser, J., and Hooper, A. N.: Medical Research Committee, Special Report Series, no. 25, 1919, p. 72.

11. Whipple, G. H.; Smith, H. P., and Belt, A. E.: Am. J. Physiol. **52**:72, 1920.

regularly by the pathologists. None of these observers commented on the significance of these visible evidences or emphasized their etiologic relationship to the mechanism by which shock results. Moon and Kennedy [12] produced shock by various methods in dogs and found regularly the same changes as described by Heubner. Recently one of us (Dr. Moon [13]) summarized as follows the changes accompanying the shock syndrome variously produced:

> The veins in subcutaneous and peripheral areas are collapsed and relatively bloodless. The viscera are deeply congested and have a purplish, diffuse, cyanotic color. This is especially prominent in the lungs, liver and gastro-intestinal tract. The venules of the visceral peritoneum are engorged and distended. Those along the mesenteric attachment are especially prominent. Usually the spleen is contracted and bloodless, though occasionally it also is engorged. There are varying quantities of thin, blood-tinged fluid in the peritoneal, pleural and sometimes in the pericardial cavities. The mucosae of the respiratory and gastro-intestinal tracts are swollen and have the appearance of red velvet. There are numerous petechial hemorrhages in the lungs, in the pleural and pericardial surfaces, in the intestinal mucosa, occasionally in the lining of the gall and urinary bladders and in the meninges. Edema of the lungs and mucosae and serous effusions are most marked when shock develops gradually and death is not immediate. When shock ending in death develops rapidly, congestion and capillary hemorrhages are marked and edema is a less prominent feature. The fluid present in the stomach usually contains "coffee-grounds" flocculi which give a positive test for hemoglobin. The fluid in the stomach and bowel contains quantities of serum albumin. The undiluted fluid often forms a solid coagulum when heated.
>
> On microscopic examination there are marked dilatation and engorgement of the capillaries and venules in each of the organs mentioned. There are numerous minute areas of hemorrhage, apparently from dissolution of capillary walls, and tissue edema is present in varying degrees. The adrenals participate in these circulatory changes in a degree similar to that seen in other organs, but they show no other changes.
>
> These observations indicate a widespread loss of tonus affecting the capillaries and venules. The circulatory disparity in this instance results from an increased volume-capacity of the vascular system or, what is equivalent, from inability of the vascular system to contract with its normal tonus on the contained blood.
>
> There was evidence that the minute vessels were abnormally permeable to blood plasma. This gave rise to edema, serous effusions, and to the transudation of fluid from the blood into the gastro-intestinal canal. Such loss of fluid lowers the blood volume and produces hemoconcentration.

The fact that the spleen usually does not share in the visceral congestion is significant. The spleen acts as a reservoir for blood not needed by the system. Shock is similar to hemorrhage in its effects, and the spleen discharges its reserve of blood in a physiologic effort to meet

12. Moon, V. H., and Kennedy, P. J.: Pathology of Shock, Arch. Path. **14**: 360 (Sept.) 1932.

13. Moon, V. H.: Shock Syndrome in Medicine and Surgery, Ann. Int. Med **8**:1633 (June) 1935.

the systemic demand. In a few instances we have found the spleen relaxed, atonic and engorged, but usually it is contracted, dry and bloodless.

In the reported experiments on intestinal obstruction the details of postmortem observations are fragmentary. Yet the records agree in the frequent mention of marked congestion and capillary hemorrhages in the gastro-intestinal mucosae in parts not involved by the obstruction. The capillaries and venules along the mesenteric attachment are engorged and distended, and there is marked congestion of other viscera, especially of the liver and kidneys. The blood clots slowly and appears concentrated. Marked congestion of the lungs and of the mucosa of the respiratory tract are reported frequently.

The decrease in blood chlorides accompanying intestinal obstruction is much discussed. This feature is not peculiar to intestinal obstruction. It follows injection of histamine in animals (Drake and Tisdall,[14] Hashimoto[15]) and is a prominent feature in shock resulting from insufficiency of the adrenal cortex (Banting and Gairns[16]). The blood chlorides are decreased in persons with many severe illnesses, including venous stasis, diabetic intoxication, pneumonia, pleurisy, burns, mercuric poisoning, eclampsia and other toxemias of pregnancy, anaphylactic shock, serum disease, etc. (Peters and Van Slyke[17]). It is of interest to note that these conditions frequently produce shock, with hemoconcentration and characteristic postmortem appearances (Atchley,[18] Eppinger,[19] Moon[20]). A decrease in blood chlorides is not a distinctive feature of intestinal obstruction but is a feature in common with other conditions terminating in circulatory disturbances of the shock type. The same statement applies with equal force to the high nonprotein nitrogen content of the blood. It also is found regularly in shock from diverse causes.

POSTMORTEM OBSERVATIONS ON ANIMALS WITH INTESTINAL OBSTRUCTION

Experimental obstructions were made in dogs to provide material for observations on the associated pathologic changes. With the animal under ether anesthesia and with aseptic technic, a tape was tied about a loop of bowel immediately below the mesenteric root. The included length of bowel ranged from 8 to 36 inches

14. Drake, T. G. H., and Tisdall, F. F.: J. Biol. Chem. **67**:91, 1926.
15. Hashimoto, H.: J. Pharmacol & Exper. Therap. **25**:381, 1925.
16. Banting, F. G., and Gairns, S.: Am. J. Physiol. **77**:100, 1926.
17. Peters, J. P., and Van Slyke, D. D.: Quantitative Clinical Chemistry, Baltimore, Williams & Wilkins Company, 1931.
18. Atchley, D. W.: Medical Shock, J. A. M. A. **95**:385 (Aug. 9) 1930.
19. Eppinger, Hans: Wien. klin. Wchnschr. **47**:10 and 355, 1919.
20. Moon, V. H.: Das Schocksyndrom, Deutsche med. Wchnschr. **60**:1667 (Nov. 2) 1934.

(20 to 90 cm.) in different dogs, and the tape was sufficiently tight to obstruct the lumen. No kymographic records were made since it was desired to avoid the complicating effects of prolonged anesthesia, which often produces dilatation and engorgement of minute vessels, resulting in shocklike phenomena. Such effects are usually present if deep narcosis with one of the barbiturates has been employed. Red cell counts and estimations of hemoglobin were made before and at intervals following the operations. Hemoconcentration served in these as in previous experiments to indicate the development of circulatory failure. A few representative protocols of these experiments follow:

Dog 170.—This was a mongrel hound weighing 25 pounds (11.3 Kg.). The red cell count was 6,800,000, the pulse rate 100, the respiratory rate 56 and the temperature 101.4 F.

On February 5, at 8:00 a. m., with the animal under ether anesthesia a loop at the upper end of the jejunum 8 inches (20 cm.) in length was ligated with tape. The dog had marked tremors, was listless and inactive and vomited occasionally.

Fig. 1.—Photograph showing congestion of the lungs of dog 170 compared with those of a normal dog (left).

At 4:30 p. m. the red cell count was 8,890,000, the pulse rate 170, the respiratory rate 40 and the temperature 101.8 F. On February 6, at 8:00 a. m., the dog was found in his cage dead and cold.

Postmortem Examination.—The lungs, pleura and pericardium were intensely congested (fig. 1). There was no free fluid in the chest cavities. There was 125 cc. of blood-tinged fluid in the peritoneal cavity. The peritoneum was markedly congested throughout but showed no evidence of peritonitis. The venules along the mesenteric attachment were distended and engorged. The ligated loop of bowel was distended, intensely hemorrhagic and very dark. The peritoneum covering the entire bowel tract was reddish purple. The mucosa of the bowel, both above and below the obstruction, was intensely congested and diffusely hemorrhagic (fig. 2). The mucosa of the stomach was greatly congested and purple. There was marked congestion of the liver and kidneys. The spleen was dry, contracted and bloodless. The adrenal glands appeared normal.

Histologic Examination: In the lung there was moderate congestion of the capillaries and venules and moderate edema of the alveoli and bronchial mucosa. There were scattered capillary hemorrhages. The mucosa of the unobstructed bowel showed marked congestion, slight edema and capillary hemorrhages. The venules and capillaries of the papillae were markedly distended and tortuous (fig. 3). There was moderate congestion of the mucosa of the stomach. The pulp of the spleen was anemic. In the kidney there were marked congestion, edema, capillary hemorrhages and marked parenchymatous degeneration. The liver showed moderate congestion of the sinusoids. The heart muscle, adrenal glands and pancreas showed no changes.

Dog 171.—This was a mongrel weighing 21 pounds (9.5 Kg.). The red cell count was 8,000,000, the pulse rate 80, the respiratory rate 40 and the temperature 103 F.

On February 6, at 8:00 a. m., about 3 feet (90 cm.) of the upper part of the small bowel was included in a loop ligated with tape. At 1:30 p. m. examination revealed that the red cell count was 9,600,000, the pulse rate 108, the respiratory

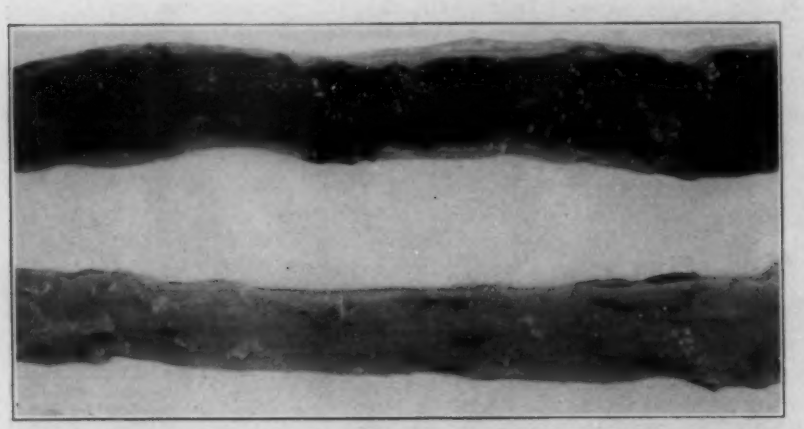

Fig. 2.—Photograph showing congestion and hemorrhage of the mucosa of the unobstructed portion of the bowel of dog 170 compared with the small bowel from a normal dog (below).

rate 48 and the temperature 103.5 F. The dog was inactive and listless and vomited frequently. At 2:30 p. m., 10 cc. of a 1 per cent neutral solution of trypan blue was given intravenously. At 7:30 p. m., the dog was comatose and moribund. The red cell count was 9,980,000, the pulse rate, 120, the respiratory rate 40 and the temperature 102.5 F.

Postmortem Examination.—There was no fluid in the pleural cavities. The pleural surfaces of the lungs contained petechial hemorrhages. There was moderate congestion of the lung substance. There were petechial hemorrhages and marked bluing of the endocardial surfaces. The obstructed loop of bowel was greatly distended and intensely hemorrhagic. The peritoneal cavity contained about 150 cc. of thin blood-tinged fluid. There was no peritonitis. The entire gastro-intestinal mucosa above and below the obstructed loop was intensely colored with the trypan blue. It contained numerous ecchymoses and was swollen and edematous. The liver and kidneys were congested and mottled with hemorrhagic areas. The kidneys and bladder showed marked staining with the trypan blue.

There was also staining of the mesentery and of the retroperitoneal lymph nodes. The adrenal glands were slightly congested but otherwise appeared normal.

Histologic Examination: In the lung there were intense congestion of the venules and capillaries, numerous capillary hemorrhages and beginning edema. The mucosa of the unobstructed bowel was intensely congested and contained numerous capillary hemorrhages. The stomach showed marked congestion and capillary hemorrhages in the mucosa. In the kidney there were marked congestion, numerous capillary hemorrhages and marked parenchymatous degeneration. In the liver there were congestion of sinusoids and parenchymatous degeneration. The heart muscle, pancreas and adrenal glands were normal.

Dog 173.—This was a male mongrel weighing 22 pounds (10 Kg.). The red cell count was 6,350,000, the pulse rate 64, the respiratory rate 29 and the temperature 103.2 F.

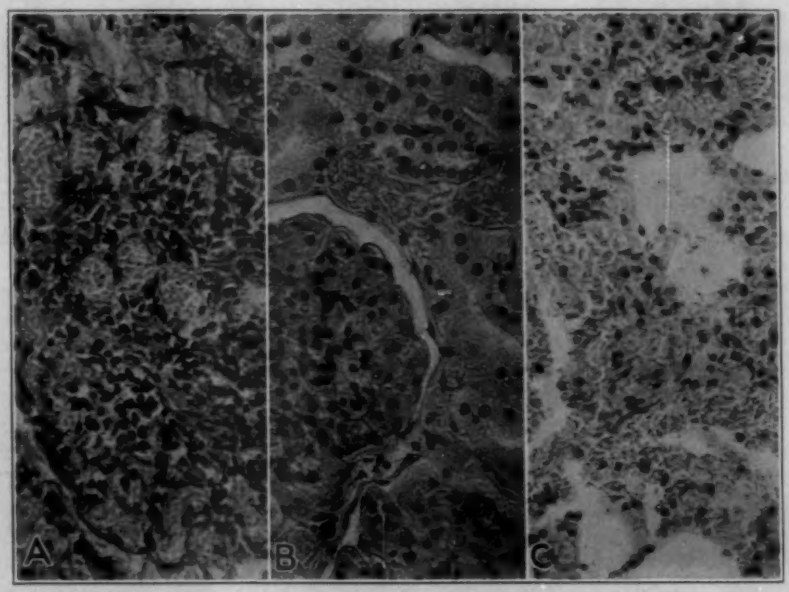

Fig. 3.—Photomicrographs of mucosa of the unobstructed bowel (*A*), the kidney (*B*) and the lung (*C*) from a dog with intestinal obstruction at a high level. Note the edema and the marked distention of the papillary venules of the mucosa. The glomerular and intertubular capillaries are congested, and there are congestion and capillary hemorrhages in the lung.

On February 13, at 4:00 p. m., a loop of the intestine approximately 10 inches (25 cm.) in length was ligated with tape. The duration of the operation was ten minutes. On February 14, at 8:00 a. m., the red cell count was 8,880,000, the pulse rate 85, the respiratory rate 25 and the temperature 103 F. The dog showed the usual evidences of illness, including muscular tremors, frequent vomiting, rapid pulse, weakness and apathy. At 5:00 p. m., the red cell count was 10,000,000, the pulse rate 90, the respiratory rate 30 and the temperature 102.7 F. Ten cubic centimeters of a 1 per cent neutral solution of trypan blue was injected intravenously. At 9:00 p. m., the dog was semicomatose and apparently moribund. The red cell count was 10,900,000, the pulse rate 140, the respiratory rate 18 and the temperature 103.8 F.

Postmortem Examination.—The visceral and parietal pleurae were greatly congested and were stained with trypan blue. About 100 cc. of blood-tinged bluish fluid was in each pleural cavity. There were petechial hemorrhages in the pleura and in the lung substance. The pericardium was congested, contained petechial hemorrhages and was moderately stained with blue. There was intense blue staining of the parietal wall and of the visceral peritoneum. There were marked congestion and bluing of the mucosa of the stomach. The obstructed portion of the bowel was intensely hemorrhagic and distended. The internal and external surfaces of the unobstructed bowel were greatly congested, edematous and intensely stained with blue. The peritoneal cavity contained about 150 cc. of blood-tinged fluid. There was no peritonitis. The liver and kidneys were markedly congested. The kidneys were stained markedly with blue. The lining of the bladder was moderately congested and stained with blue.

Microscopic Examination: In the lung there were diffuse congestion, scattered capillary hemorrhages and slight edema. The pulp of the spleen was anemic. In the small bowel there were marked congestion, edema and capillary hemorrhages of the mucosa. There was moderate congestion of the mucosa of the stomach. The kidney showed marked congestion and parenchymatous degeneration. There was slight congestion of the liver. In the bladder there was marked congestion of the venules and capillaries of the lining. The heart muscle, skeletal muscle and adrenal glands showed no visible change.

Experiments on Other Dogs.—Six other dogs, 174 to 179 inclusive, were treated in exactly the same fashion and with exactly similar results, except that in a few instances trypan blue was not given. The evidences of illness and the gross and microscopic changes were not different in any particular from those reported in the protocols of the previous experiments. The time elapsing between the operation and death ranged from sixteen to thirty hours. These dogs did not die of peritonitis. They showed the same characteristics as are seen in the shock syndrome produced by various means.

In another experiment injections of aqueous extracts of intestinal mucosa were tested for their effects on blood pressure. The fresh duodenum and jejunum from a normal dog were opened and washed free from their contents. The mucosa was scraped with a dull knife, and one volume of the scrapings was suspended and vigorously shaken with four volumes of physiologic solution of sodium chloride. This was centrifugated at high speed for ten minutes, and the clear supernatant fluid was injected intravenously into dogs which had been anesthetized with ether and had a cannula in the carotid artery recording blood pressure through a mercury manometer. An aqueous extract of mucosa from an obstructed loop of bowel was prepared and used in exactly the same manner. The mucosa from such obstructed loops is diffusely hemorrhagic and partially necrotic and contains many bacteria. Hence, as may be expected, its effects are more severe.

Dog 180 was given 10 cc. of extract of normal mucosa. The blood pressure promptly fell 56 mm. and then gradually returned to normal. In dog 181 and 182 the blood pressure fell 70 mm. and 62 mm., respectively, following the injection of the same volume of extract. In each instance the blood pressure returned to normal within thirty minutes. At subsequent postmortem examinations there was slight diffuse congestion of the lungs, gastro-intestinal mucosa and liver. No other significant changes were present.

Dog 183 received 10 cc. of extract of mucosa from an obstructed loop. This produced a rapid fall in blood pressure ending in death almost immediately. Dog

184 received the same dose, with a similar result. Dog 185 was given 5 cc. of the same extract. This produced an immediate fall in blood pressure ending in death in ten minutes. At postmortem examination there was marked diffuse congestion of the lungs, bronchial mucosa, intestinal mucosa, liver and kidneys. The venules along the mesenteric attachment were engorged and prominent. The adrenal glands were slightly congested. The spleens were contracted and bloodless. Microscopically there were marked congestion and numerous capillary hemorrhages in the lungs and gastro-intestinal mucosae. No edema was present.

This experiment illustrates the fact that extracts of normal tissues, in this instance intestinal mucosa, produce a fall in blood pressure. Whether this is due to histamine or to products of protein cleavage produced by autolysis is not apparent. Probably it is due to the combined effects of both. The extract of mucosa from the obstructed bowel would probably not contain more histamine than normal mucosa; but it would contain more products of necrotic disintegrating tissue. The observation is not new that extracts of disintegrating necrotic tissues are toxic and will produce a fall in blood pressure and other manifestations of shock if introduced into the circulation. In our experiment the extract of obstructed mucosa was much more potent that that of normal mucosa under like conditions.

It is probable that the absorption of substances from strangulated loops takes place in part via the blood and lymph channels if these are not obstructed and in part by diffusion into the peritoneal cavity with absorption through the peritoneal surfaces. The products of bacterial growth will be factors of importance in proportion to their amount and toxicity. The combined effects of histamine, products of disintegration of tissue and products of bacterial activity, any one of which alone will produce shock, will account for the clinical manifestations and postmortem observations in cases of intestinal obstruction or strangulation, whether occurring spontaneously or experimentally.

It has been noted repeatedly that the effects of simple obstruction are much less marked than those of strangulation. Apparently the mechanism is similar, and the difference is quantitative rather than qualitative.

INTESTINAL OBSTRUCTION IN HUMAN BEINGS

In a survey of the records of postmortem examinations of the past ten years in Jefferson Hospital, twenty-six cases were found in which intestinal obstruction was a chief or a contributory cause of death. The obstructions were of various degrees, types and locations, and the duration of illness varied. Peritonitis was a complicating factor in many cases. The cases in which obstruction was a major factor had the usual clinical characteristics. Such features as terminal circulatory failure, with rapid weak pulse, falling blood pressure and pulmonary congestion and edema, were prominent in the clinical histories. The majority of

these necropsies were made before it was recognized that visible circulatory changes regularly accompany the shock syndrome. Such changes as marked visceral congestion, edema of mucosae and of the lungs, capillary hemorrhages in mucous and serous surfaces and blood-tinged serous effusions were noted frequently in these postmortem records, though without comment on their significance. The following case is illustrative:

A man, aged 54, became weak and sick while working. He had abdominal pain and an urgent desire to defecate. Nausea and vomiting developed, and there was frequent passage of watery feces. The abdomen was somewhat distended and tender, but there was no rigidity. Tympany and active peristalsis were noted. On the following day, the bowel movements had ceased, but the other symptoms increased in severity. The skin was cold and clammy, the nail beds and lips became cyanotic and the temperature was subnormal. The blood pressure was 116 systolic and 88 diastolic, and the radial pulse rate was 114. The red cell count was not recorded. The vomitus was voluminous and contained visible blood. A stomach tube was introduced, and large quantities of fluid were removed from the stomach. Moderate congestion and edema of the lungs were noted. Intravenous injections of dextrose in saline solution and stimulants were given without benefit. On the next morning, the blood pressure was not measurable. The radial pulse was very rapid and barely palpable. The patient became comatose and died about sixty-five hours after the onset of illness. The chief of staff noted on the record: "This man was in a state of shock."

At necropsy a loop of ileum about 7 feet (213 cm.) long was found obstructed by adhesions. The mucosa of this was markedly hemorrhagic and contained areas of necrosis. Thrombi were present in the small mesenteric vessels. The fluid from the stomach contained "coffee-ground" flocculi. The mucosa of the stomach and that of the unobstructed bowel were swollen, deeply congested and ecchymotic. The peritoneal cavity contained blood-tinged fluid, but there was no evidence of generalized peritonitis. The lungs were markedly congested, had increased in weight and contained numerous small hemorrhagic areas. Moderate pulmonary edema was present. The liver and kidneys were congested. The spleen was not congested but was tough and flabby in consistency. The adrenal glands showed no visible changes.

Details of other cases of this type would be a repetition of observations well established and recognized by surgeons. The changes observed post mortem do not differ from those usually described. They are those which regularly accompany the shock syndrome, whether it occurs clinically or as a result of experimentation.

COMMENT

The shock syndrome is a grave circulatory disturbance, which may originate in different ways. It arises whenever there is a serious disparity between the volume of blood and the volume capacity of the vascular system. Such a disparity may develop from a decreased volume of blood, from an increased volume capacity of the vascular system or

from combinations of these. In human patients with shock and in animals with experimentally induced shock there is visible postmortem evidence of an increased volume capacity of the vascular system. This is seen in the abnormal dilatation of the venules and capillaries. A decreased volume of blood may result from loss of plasma by transudation through capillary walls. Evidence of this is seen in the edema of mucosae, in the effusions of serum into cavities lined with serous membrane and pulmonary alveoli and in the numerous capillary hemorrhages occurring in mucous and serous surfaces and in the parenchyma of organs.

When capillaries are injured, by whatever means, the permeability of their walls is increased (Landis [21]). Injury to the capillaries may result from a wide variety of agents. These include various chemical poisons, such as mercury bichloride, arsenical compounds, gold salts, alcohol and anesthetic drugs, such as barbital. Histamine, peptone, products of protein cleavage and extracts of various tissues cause injury to the capillaries; deficiency of oxygen of moderate degree and short duration will cause capillaries to dilate and their permeability to be increased (Krogh,[22] Landis [21] and others).

When permeability of the capillaries is increased in extensive areas, the transudation of plasma through their walls causes edema in the tissues and increases the concentration of the blood. The dilated capillaries and venules are filled with viscid concentrated blood, and the heart is unable to maintain normal blood pressure because of insufficient return of blood from the periphery. The heart is not a suction pump; it cannot draw blood out of the tissues but can only pump the blood which is returned to it. As the circulation becomes ineffective, lack of oxygen contributes to further capillary atony, and the circulatory disturbance becomes more grave (Cannon [10]).

If lowered blood volume and hemoconcentration are produced by plasmapheresis without injury to the capillaries the animal may return to normal spontaneously or following the therapeutic administration of fluid. Fluid is absorbed if given by mouth or by hypodermoclysis, and fluids injected intravenously are retained in the circulation. In shock fluids are neither absorbed nor retained. Injection of them into the blood merely increases the edema. It appears that an essential factor by which shock develops and progresses is increased permeability of the capillaries.

Many workers have used colloidal dyes as indicators of permeability of the capillaries. Such dyes as trypan blue do not pass readily through

21. Landis, E. M.: Physiol. Rev. **14**:404, 1934.

22. Krogh, A.: The Anatomy and Physiology of Capillaries, ed. 2, New Haven, Yale University Press, 1929.

normal capillary walls but will permeate the tissue spaces where permeability of the capillaries is increased. In our experiments there was marked staining of the serous and mucous surfaces, kidneys, bladder and other tissue in animals brought to death by intestinal obstruction. This observation supplied visible evidence of increased permeability of the capillaries in extensive areas.

The hemoconcentration accompanying intestinal obstruction has been assigned to loss of fluid by vomiting. Such fluid comes from the blood, largely because of permeability of the capillaries. The fluid found in the stomach and frequently that in the bowel contain quantities of albumin. The "coffee-ground" vomitus from such patients contains hemoglobin and serum albumin which have escaped through the walls of the capillaries. Such loss of fluid is an important factor contributing to decreased blood volume and to hemoconcentration. The effusions into serous cavities and the edema of the lungs and mucosae are likewise important factors. The combined effect of loss of blood volume by transudation and of increased volume capacity due to atony of capillaries and venules produces a circulatory insufficiency which manifests itself in the shock syndrome.

CONCLUSIONS

There is much evidence that death following intestinal obstruction results from circulatory failure of the shock type.

The physiologic disturbances following intestinal obstruction are those usually seen in the shock syndrome.

The gross and microscopic visceral changes are identical with those which accompany shock produced by various means. These changes are: marked distention and engorgement of the capillaries and venules in the viscera, edema and ecchymoses of the lungs and gastro-intestinal mucosae and effusions into serous cavities. Evidence of injury to the capillaries is seen in the capillary hemorrhages, the formation of edema fluid of high protein content and the rapidity with which colloidal dyes escape from the blood into the tissues.

The probable cause of the shock syndrome following intestinal obstruction is intoxication by histamine, by other products of injured tissue and by bacteria and their products absorbed from the obstructed bowel. Any of these agents alone will produce the shock syndrome in animals. Debate concerning the relative clinical importance of these agents is not profitable.

Intestinal obstruction is one of the many conditions which will produce characteristically the shock syndrome.

REGIONAL ILEITIS

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In the past few years a number of papers dealing with the question of nonspecific inflammatory lesions in the gastro-intestinal tract have appeared. Some of the papers dealt with the subject at length. Others reported only a few cases with comments. A great deal remains to be clarified, especially regarding the early phases of the disease. More specific information is needed relating to (1) prognosis as to life, (2) incidence of recurrence even after radical surgical intervention, and (3) the end-results in cases of the early stages noted at operation, in which no resection or other intestinal procedure is done. Criteria for clinical diagnosis must be established, as it becomes more obvious daily that the condition is not uncommon and must surely be noted from time to time in the routine experience of the general practitioner. He must be prepared to recognize the syndrome or at least the possibilities thereof. He should know what diagnostic steps to take in an attempt to answer the foregoing questions as intelligently as is possible. With these needs in mind we have reviewed the publications on intestinal granuloma, studied the reports of cases and tabulated the clinical facts derived therefrom. To this survey we have added observations on 17 cases of our own, which we shall discuss in greater detail.

From July 14, 1931, to Dec. 6, 1934, we encountered 17 instances of a peculiar inflammatory lesion localized in the terminal portion of the ileum, ranging in severity from the mildest type of inflammatory process to the severest type of deformation of the wall and lumen of the intestine, with ulceration and, in some cases, fistulas following perforation. These cases we believe can properly be considered under the heading of nonspecific inflammatory disease of the intestinal tract, previously described by many authors. Although the lesion was recognized many years ago, it is only within the last few years that interest in it has been stimulated by the appearance of reports of the lesion in the small intestine.

A synopsis of the histories of the 17 cases is given in table 1. In table 2, an analysis of these cases is presented.

TABLE 1.—*Observations on 17 Personal*

	Date of Admission	Age and Sex	Previous Operations	Symptoms on Admission	Duration of Symptoms	Physical Findings	Preoperative Diagnosis	Laboratory Data
1	7/14/31	23 M	Appendectomy 4 yr. before, followed by fecal fistula; three attempts to close fistula unsuccessful	Fecal discharge from abdominal wound; loss of 40 lb. (wt. 80 lb.); abdominal cramps; general weakness; T., 99 F.; P. R., 80; R. R., 20	4½ yr.	Debilitated; 3 scars in lower right quadrant with fecal fistula at lower end of a McBurney scar; entire abdomen very tender; lungs normal	Fecal fistula of tuberculous nature	Hem. 77%; R.B.C. 4,250,000; W.B.C. 10,000; poly. 77%; mono. 7%; lymph. 16%; urine and feces normal
2	7/ 5/32	26 M	None	Pains in right lower quadrant with nausea and vomiting; no previous attack; T. 100.6 F.; P. R. 90; R. R. 22	3 da.	Well developed; acutely ill; tenderness, rigidity with rebound tenderness in right lower quadrant with slight distention	Acute appendicitis	Hem. 92%; R.B.C. 4,800,000; W.B.C. 18,200; poly. 92%; lymph. 8%; urine normal
3	9/ 4/32	16 F	None	Pains in right lower quadrant with nausea and vomiting; 2 previous attacks; T. 99.2 F.; P. R. 100; R. R. 22	3 da.	Weight 90 lb.; tenderness, rigidity with rebound tenderness in right lower quadrant with mass palpable; moderate distention	Acute appendicitis	Hem. 75%; R.B.C. 3,900,000; W.B.C. 6,700; poly. 60%; lymph. 29%; mono. 11%; urine normal
4	9/13/32	24 F	None	Pains in right lower quadrant; vomiting; no previous attack; T. 100.8 F.; P. R. 112; R. R. 24	4 da.	Well developed; tenderness, rigidity with rebound tenderness in right lower quadrant with moderate distention	Acute appendicitis	Hem. 80%; R.B.C. 3,965,000; W.B.C. 20,100; poly. 94%; lymph. 3%; mono. 3%; urine normal
5	11/13/32	34 M	None	Pains in right lower quadrant with nausea and vomiting; no previous attack; T. 102.2 F.; P. R. 108; R. R. 22	2 da.	Well developed; tenderness, rigidity with rebound tenderness in right lower quadrant; no distention	Acute appendicitis	Hem. 100%; R.B.C. 5,000,000; W.B.C. 16,200; poly. 90%; lymph. 10%; urine normal
6	11/15/32	14 F	None	Pains in right lower quadrant with nausea and vomiting; no previous attack; T. 100 F.; P. R. 110; R. R. 22	6 da.	Well developed; tenderness, rigidity of entire lower quadrant with marked rebound tenderness; slight distention	Acute appendicitis	Hem. 99%; R.B.C. 4,980,000; W.B.C. 27,000; poly. 87%; lymph. 10%; mono. 3%; urine normal
7	7/30/33	28 M	None	Pains in right lower quadrant and epigastrium for past year, with nausea and vomiting at times; T. 99.4 F.; P. R. 70; R. R. 20	1 yr.	Well developed; marked tenderness in right lower quadrant with hyperesthesia; roentgen examination showed pathologic changes in appendix	Chronic appendicitis	Hem. 100%; R.B.C. 4,930,000; W.B.C. 11,300; poly. 68%; lymph. 28%; mono. 4%; urine normal
8	11/ 2/33	35 M	None	Intermittent pains about umbilicus with nausea and constipation; loss of 10 lb. in 6 mo.; T. 99.8 F.; P. R. 84; R. R. 24	3 yr.	Well developed; tender and distended abdomen with visible peristalsis; no definite mass palpable; enema resulted in no return and increased pains	Partial intestinal obstruction	Hem. 100%; R.B.C. 5,130,000; W.B.C. 13,600; poly. 67%; lymph. 30%; mono. 3%; urine normal

e	Laboratory Data	Observations at Operation	Surgical Procedure	Pathologic Reports	Postoperative Course	Discharge	Present Status	Comment
	Hem. 77%; R.B.C. 4,250,000; W.B.C. 10,600; poly. 77%; mono. 7%; lymph. 16%; urine and feces normal	gmoid flexure attached to cecum and rminal ileum, with ultiple fistulous acts and sinuses ptying into one large act to opening in bdominal wall; ileostomy to ascending colon	Sigmoid separated from cecum; opening in sigmoid closed by purse-string suture; entire ileocecal region excised, including fistulous tracts and mesentery; 3 layered closure of open ends of intestine; cigaret drain to abdomen	Chronic ulcerating ileitis of nonspecific inflammatory origin; tuberculosis definitely ruled out	Drain removed on fifth day; sutures removed on eighth day; uneventful recovery	8/31/31	11/30/34: wt. 140 lb.; bowel movement regular; no complaint	Weight at discharge, 116 lb.; operation subsequently for ischiorectal fistula with good result
	Hem. 98%; R.B.C. 4,300,000; W.B.C. 18,200; poly. 98%; lymph. 8%; urine normal	ppendix injected; rge amount of serous id; about 1 ft. from ecal junction, 3 ft. ileum involved in flammatory thickening with edema of its esentery and enlarged ands; partial estruction	Resection of involved ileum, including its mesentery and glands, with an end-to-end ileo-ileostomy; smears for culture taken from abdomen; no drainage	Chronic nonspecific inflammation of ileum; cultures of abdominal fluid negative	Sutures removed on eighth day; uneventful recovery	7/19/32	12/3/34: no complaint; bowel movement daily; gain of 16 lb.	Acute symptoms due to obstruction caused by the stenosing lesion
	Hem. 75%; R.B.C. 3,300,000; W.B.C. 6,700; poly. 60%; lymph. 29%; mono. 11%; urine normal	larged mesenteric ands; inflammatory ass of ileum, cecum d omentum with appendix normal but adherent to congested, ickened ileum; rforation of um near ileocecal nction walled off by entum, with almost mplete obstruction	Resection of terminal 9 in. of ileum and cecum, including enlarged glands, with side-to-side ileocolostomy to ascending colon; no drainage	Chronic nonspecific inflammation of ileum with superimposed subacute ulcerating lesions	Sutures removed on seventh day; uneventful recovery	9/17/34	10/31/34: no complaint; x-rays showed well functioning stoma, wt. 122 lb.	Weight at discharge, 102 lb.; this case typifies ulcerating nature of lesion
	Hem. 80%; R.B.C. 3,955,000; W.B.C. 20,100; poly. 94%; lymph. 3%; mono. 3%; urine normal	ee serous fluid in abmen; appendix conested and adherent to ass consisting of um, cecum, right tube d ovary, omentum; rminal ileum enlarged, flamed and thickened, ith obstruction in ecal region	Resection of terminal 14 in. of ileum and cecum with end-to-side ileocolostomy to ascending colon; cigaret drain inserted to abdomen	Chronic nonspecific inflammation of ileum with stenosis of ileocecal valve	Drain removed on fifth day; sutures removed on seventh day; uneventful recovery	9/26/32	10/31/34: bowel movement regular; no complaint; x-rays showed good stoma; gain of 10 lb.	Acute symptoms due to obstruction at ileocecal valve
	Hem. 100%; R.B.C. 5,000,000; W.B.C. 16,200; poly. 90%; lymph. 10%; urine normal	ee serous fluid, appendix congested; terminal 12 in. of ileum flamed, slightly thickened, with edema of esentery and enlarged ands; no obstruction	Appendectomy; insertion of cigaret drain into abdomen because of large amount of fluid; one mesenteric gland removed for biopsy	No lesion of appendix; acute nonspecific inflammation of lymph node	Drain removed on fifth day; sutures removed on eighth day; uneventful recovery	11/25/32	11/13/34: no complaint; bowel movement regular; gain of 14 lb.	Early stage of regional ileitis with acute symptoms
	Hem. 99%; R.B.C. 4,900,000; W.B.C. 27,000; poly. 87%; lymph. 10%; mono. 3%; urine normal	rge amount of serohemorrhagic fluid; inted appendix; hemrrhagic, markedly ickened, 14 in. of um, beginning 6 in. om ileocecal junction; mesentery ematous; glands larged	Resection of involved ileum, including its mesentery and glands, with end-to-end ileo-ileostomy; appendectomy; no drainage	No lesion of appendix; chronic nonspecific ileitis, with superimposed acute purulent inflammation, areas of necrosis and ulceration	Sutures removed on seventh day; uneventful recovery	12/3/32	11/10/34: bowel movement regular; no complaint; gain of 24 lb.	Ulcerating stage of regional ileitis provoking marked peritoneal reaction
	Hem. 100%; R.B.C. 4,950,000; W.B.C. 11,200; poly. 68%; lymph. 28%; mono. 4%; urine normal	ight amount of rous fluid; inflammaon and slight thickning of about 4 in. of um about 1 ft. from ecal junction; derate edema and larged glands in e vicinity of the volved ileum	Appendectomy; excision of one mesenteric gland for biopsy; no drainage	Chronic appendicitis; chronic nonspecific inflammation of lymph node	Sutures removed on seventh day; uneventful recovery	8/12/33	12/30/33: no complaint; bowel movement regular; gain of 11 lb.	Early stage of regional ileitis; high voltage roentgen therapy to abdomen in 2 doses one week after leaving hospital
	Hem. 100%; R.B.C. 5,150,000; W.B.C. 13,600; poly. 67%; lymph. 30%; mono. 3%; urine normal	derate amount of rous fluid; marked ickening, inflammaon of terminal 10 in. ileum; ileocecal nds enlarged and flamed	Ileocolostomy of ileum 2½ ft. from ileocecal valve to transverse colon; no drainage; (11/3/33)	Chronic nonspecific ileitis, with marked hypertrophy of the musculature	Sutures removed on seventh day; uneventful recovery	11/26/33	11/30/34: bowel movement regular; no complaint; gain of 18 lb.	Two stage resection performed because of acute obstruction and patient's general condition
		second laparotomy, dings same; well nctioning ileoic stoma	Resection of terminal 15 in. of ileum, cecum and half of ascending colon, with closure of open ends; no drainage (11/14/33)		Sutures removed on eighth day; uneventful recovery			

TABLE 1.—*Observations on 17 Persons*

	Date of Admission	Age and Sex	Previous Operations	Symptoms on Admission	Duration of Symptoms	Physical Findings	Preoperative Diagnosis	Laboratory Data
9	12/18/33	7 M	None	Pains in right lower quadrant with nausea and vomiting; 1 previous attack; T. 103.6 F.; P. R. 130; R. R. 24	2 da.	Well developed; tenderness, rigidity with rebound tenderness in right lower quadrant	Acute appendicitis	Hem. 102%; R.B.C. 5,100,000; W.B.C. 19,800; poly. 89%; lymph. 4%; mono. 7%; urine normal
10	1/18/32 (1st adm.)	37 F	None	Pains in right lower quadrant with nausea and vomiting; constipation; several previous attacks in past year; T. 98.6 F.; P. R. 120; R. R. 28	2 wk.	Well developed; tenderness and rigidity with rebound tenderness in right lower quadrant; moderate distention	Acute appendicitis	Hem. 98%; R.B.C. 4,200,000; W.B.C. 15,800; poly. 85%; lymph. 12%; mono. 3%; urine normal
	2/12/34 (2d adm.)		Ileocolostomy 25 days before	Diffuse abdominal cramps and vomiting; T. 99.8 F.; P. R. 100; R. R. 24		Marked tenderness in right lower quadrant; moderate distention		W.B.C. 18,300; poly. 87%; lymph. 13%
11	5/10/34	21 F	None	Pains in right lower quadrant with nausea and vomiting; 3 attacks in past 4 yr.; T. 100.8 F.; P. R. 80; R. R. 20	4 da.	Well developed tenderness, rigidity with rebound tenderness in right lower quadrant; slight distention	Acute appendicitis	Hem. 98%; R.B.C. 4,650,000; W.B.C. 12,800; poly. 71%; lymph. 21%; mono. 8%; urine normal
12	7/27/34	22 M	None	Pains in right lower quadrant with vomiting; 1 attack 6 mo. before; T. 99.8 F.; P. R. 90; R. R. 20	3 da.	Well developed tenderness and rigidity with rebound tenderness in right lower quadrant	Acute appendicitis	Hem. 118%; R.B.C. 5,250,000; W.B.C. 20,000; poly. 80%; lymph. 20%; urine normal
13	7/29/34	39 F	None	Pains in right lower quadrant with vomiting; no previous attacks; T. 101 F.; P. R. 96; R. R. 24	3 da.	Well developed and obese; tenderness and rigidity with rebound tenderness in right lower quadrant	Acute appendicitis	Hem. 90%; R.B.C. 4,800,000; W.B.C. 16,000; poly. 88%; lymph. 12%; urine normal
14	9/20/34	49 M	Appendectomy in 1910	Irregular sharp pains in right lower quadrant; worse in past 6 mo.; loss of 15 lb. in 2 wk.; constipation; x-ray of gastrointestinal tract normal; T. 99.2 F.; P. R. 86; R. R. 20	2 yr.	Well developed; old McBurney scar; irregular, slightly mobile, very tender doughy mass in right lower quadrant which was dull on percussion; moderate distention of abdomen	Nonspecific regional ileitis or carcinoma of cecum	Hem. 96%; R.B.C. 4,800,000; W.B.C. 7,100; poly. 69%; lymph. 24%; mono. 7%; urine normal
15	10/12/34	28 M	Appendectomy 8 mo. before, followed by fecal fistula	Fecal discharge from abdominal wound; cramps, vomiting, obstipation for 3 da. in spite of enema; T. 102 F.; P. R. 100; R. R. 28	3 da.	Well developed; poorly healed scar of right rectus muscle with fecal fistula in center; tenderness and rigidity in right lower quadrant; moderate distention with visible peristaltic waves	Acute intestinal obstruction and fecal fistula	Hem. 100%; R.B.C. 5,000,000; W.B.C. 19,800; poly. 89%; lymph. 6%; mono. 5%; urine normal
16	11/ 6/34	26 M	None	Recurrent pains in right lower quadrant for past 4 mo. accompanied by diarrhea and nausea; T. 101 F.; P. R. 110; R. R. 28	2 da.	Well developed; tenderness, rigidity with rebound tenderness in right lower quadrant	Acute appendicitis	Hem. 98%; R.B.C. 4,800,000; W.B.C. 16,300; poly. 86%; lymph. 14%; urine normal
17	1/ 7/35	30 M	None	Pains in right lower quadrant; nausea and vomiting; no previous attack; T. 99.8 F.; P. R. 88; R. R. 24	3 da.	Well developed; tenderness, rigidity with rebound tenderness in right lower quadrant; slight distention	Acute appendicitis	Hem. 100%; R.B.C. 5,000,000; W.B.C. 9,200; poly. 72%; lymph. 21%; mono. 7%; urine normal

Laboratory Data	Observations at Operation	Surgical Procedure	Pathologic Reports	Postoperative Course	Discharge	Present Status	Comment
Hem. 102%; R.B.C. 5,100,000; W.B.C. 20,800; poly. 89%; lymph. 4%; mono. 7%; urine normal	Appendix injected; slight amount of serous fluid; terminal 5 in. of ileum inflamed, slightly thickened, with enlarged inflamed glands at ileocecal angle	Appendectomy; excision of mesenteric gland for biopsy; no drainage	No lesion of appendix; acute nonspecific inflammation of lymph node	Sutures removed on seventh day; uneventful recovery	12/30/33	11/28/34: bowel movement regular; no complaint; gain of 15 lb.	Early stage of regional ileitis with acute symptoms; 2 treatments with roentgen rays while in the hospital
Hem. 98%; R.B.C. 4,200,000; W.B.C. 15,800; poly. 88%; lymph. 12%; mono. 9%; urine normal	Appendix injected and adherent; serohemorrhagic fluid; terminal 18 in. of ileum inflamed, thickened, with obstruction at ileocecal valve; enlarged mesenteric glands	Ileocolostomy of ileum 3 ft. from ileocecal valve to transverse colon; no drainage (1/18/34)	Subacute nonspecific ileitis with ulceration; chronic obliterative appendicitis	Sutures removed on eighth day; collection evacuated from wound on tenth day	2/ 1/34	11/30/34: bowel movement regular; no complaint; gain of 20 lb.; x-rays showed good stroma	Two stage resection deemed advisable in the face of acute obstruction; patient sent home after the first stage to get her in better shape for the second operation, but because of marked progression of the disease brought back sooner than planned
W.B.C. 18,200; poly. 87%; lymph. 13%	Mesenteric glands suppurating and walled off by omentum and small intestine, with necrosis and ulceration of involved ileum	Resection of terminal 18 in. of ileum, cecum and ascending colon, including suppurative glands, with closure of open ends; cigaret drain inserted (2/13/34)		Drain removed on eleventh day; wound healed slowly and drained for 4 wk.	3/19/34		
Hem. 99%; R.B.C. 4,700,000; W.B.C. 12,800; poly. 71%; lymph. 21%; mono. 8%; urine normal	Appendix injected; moderate serous fluid; terminal 4 in. of ileum inflamed, thickened, with stenosis of ileocecal valve and marked enlargement of mesenteric glands	Resection of terminal 1 ft. of ileum, cecum and portion of ascending colon, with ileocolostomy to transverse colon; no drainage	Subacute nonspecific ileitis, with ulceration and stenosis	Sutures removed on seventh day; uneventful recovery	5/27/34	10/30/34: bowel movement regular; no complaint; gain of 11 lb.	Patient's good general condition and youth favored a complete operation in one stage
Hem. 118%; R.B.C. 5,200,000; W.B.C. 20,000; poly. 80%; lymph. 20%; urine normal	Appendix injected; slight amount of serous fluid; terminal 12 in. of ileum inflamed, slightly thickened, with edema of the mesentery and enlarged glands	Appendectomy; mesenteric gland excised for biopsy	Chronic obliterating appendicitis; acute nonspecific inflammation of lymph node	Small right pulmonary infarct on fifth day, cleared up on eleventh day; sutures removed on seventh day	8/12/34	11/10/34: bowel movement regular; no complaint; gain of 6 lb.	Early stage of regional ileitis with acute symptoms
Hem. 90%; R.B.C. 4,800,000; W.B.C. 16,000; poly. 88%; lymph. 12%; urine normal	Appendix inflamed; free serous fluid; terminal 5 in. of ileum inflamed, slightly thickened, with edema of mesentery and enlarged glands	Appendectomy and culture of abdominal fluid	Subacute appendicitis; culture negative	Sutures removed on seventh day; uneventful recovery	8/15/34	12/10/34: bowel movement regular; no complaint; gain of 5 lb.; x-rays showed slight narrowing of terminal ileum	Early stage of regional ileitis with acute symptoms
Hem. 90%; R.B.C. 4,800,000; W.B.C. 7,100; poly. 60%; lymph. 24%; mono. 7%; urine normal	Terminal 2 ft. of ileum inflamed, with very thick walls and obstruction to ileocecal valve; edema of mesentery, with enlarged glands; moderate amount of serous fluid	Ileocolostomy through right rectus incision between ileum 3 ft. from ileocecal junction to transverse colon; no drainage (9/21/34)	Subacute nonspecific ileitis, with ulceration	Sutures removed on eighth day; uneventful recovery	Died 10/15/34	.....	Collection developed in lumbar wound, which was opened and drained; condition seemed to progress satisfactorily until 10/14/34, when anuria developed, which rapidly advanced to uremia and death
	Ulceration and necrosis of wall of ileum; mesenteric glands beginning to suppurate	Through incison in right kidney, resection of 2 ft. of terminal ileum, cecum and portion of ascending colon; cigaret drain (10/4/34)		Infection of wound with fascial slough			
		Blood transfusion 10/14/34					
Hem. 100%; R.B.C. 5,000,000; W.B.C. 19,800; poly. 89%; lymph. 6%; mono. 5%; urine normal	Terminal 10 in. of ileum and cecum inflamed and thickened with complete occlusion of ileocecal valve; free serous fluid; fistulous tract extending from stump of appendix to anterior abdominal wall; marked distention of proximal end of small intestine	Fistulous tract excised circumferentially down to cecum; resection of terminal 14 in. of ileum, cecum and half of ascending colon, with ileocolostomy between ileum 2½ ft. from ileocecal junction to transverse colon; Witzel enterostomy about 8 in. proximal to anastomosis; no drainage	Chronic nonspecific ileitis, with superimposed acute ulcerations; complete occlusion of ileocecal lumen	Enterostomy tube removed on seventh day; sutures removed on eighth day; uneventful recovery	10/29/34	12/13/34: bowel movement regular; no complaint; gain of 9 lb.	Operation performed in one stage in spite of acute obstruction because of patient's good general condition; Witzel enterostomy assured success of the procedure
Hem. 98%; R.B.C. 4,800,000; W.B.C. 16,800; poly. 86%; lymph. 14%; urine normal	Appendix congested; terminal 9 in. of ileum inflamed, slightly thickened; free serohemorrhagic fluid, with edema of mesentery	Appendectomy and biopsy of ileum	Subacute nonspecific ileitis, with muscular hypertrophy; chronic appendicitis	Sutures removed on seventh day; uneventful recovery	11/18/34	12/13/34: bowel movement regular; no complaint; gain of 4 lb.	Early stage of regional ileitis with acute symptoms
Hem. 100%; R.B.C. 5,000,000; W.B.C. 9,200; poly. 72%; lymph. 21%; mono. 7%; urine normal	Appendix congested; terminal 18 in. of ileum inflamed and somewhat thickened; edema of mesentery and enlarged glands; no obstruction; moderate amout of serous fluid	Appendectomy and biopsy of ileum	Acute nonspecific ileitis; chronic appendicitis	Sutures removed on eighth day; uneventful recovery	1/19/35	2/2/35: bowel movement regular; no complaint; gain of 12 lb.	Early stage of regional ileitis with acute symptoms

Our cases occurred mainly in young patients; 11 in males and 6 in females. The duration of symptoms varied from a few days to four years before operation was performed.

TABLE 2.—*A Summary of the Personally Observed Cases*

	Number of Cases
Age incidence	
1 to 10 years	1
11 to 20 years	2
21 to 30 years	8
31 to 40 years	5
49 years	1
Sex	
Males	11
Females	6
Duration of symptoms	
7 days or less	6
4 to 6 months	5
6 to 12 months	3
2, 3 and 4½ years	3
Abdominal pain	
Generalized	13
Localized in right lower quadrant	14
Diarrhea	1
Nausea	11
Vomiting (2 without nausea)	13
Previous attacks of abdominal cramps (prior to acute attacks that caused hospitalization)	10
Loss of weight (all advanced stage)	3
Previous operation (appendectomy complicated by fecal fistula 8 months before, 4 years before, 24 years before)	3
Tenderness	17
Spasticity (right lower part of abdomen)	13
Rebound tenderness	13
Distention	10
Mass (palpable on both abdominal and rectal examination)	2
Visible peristalsis	2
Fecal fistulas	2
Temperature	
Below 99 F.	1
99 to 100 F.	13
100 F. or above	3
Pulse rate	
80 or less	3
80 to 100	8
100 to 120	6
Respiratory rate (no elevation in any case)	
White blood cell count and differential	
8,000 or less	2
8,000 to 10,000	1
10,000 to 12,000	2
12,000 to 14,000	2
14,000 to 16,000	2
16,000 to 18,000	3
18,000 to 20,000	3
20,000	2
Procedures performed	
Simple appendectomy	7
One stage resection (including 2 cases of fecal fistula)	7
Two stage resection	3

The outstanding symptoms were pain in the right lower abdominal quadrant, nausea and vomiting. Physical examination revealed abdominal tenderness in all the cases, spasticity and rebound tenderness in 13 and distention in 10. Visible peristalsis was noted in 2 cases; a mass palpable on abdominal and rectal examination was encountered

in 2 cases. The details of temperature, pulse rate and variations in the white blood cell count are given in table 1. In 7 cases simple appendectomy was performed; in 7, a one stage resection, and in 3, a two stage resection. In all the cases but 1, there was a fairly consistent relationship between the extent of the lesion and the duration of symptoms.

Clinically, the condition can be divided into three stages. The first stage is characterized by a mild acute inflammation of the wall, with edema noticeable, particularly at the mesenteric border of the intestine. The second stage is one in which the ulceration and the deposition of fibrous tissue in the wall have progressed to such a degree that restitution to normal is not possible. With a continuation of the inflammatory process, either stenosis or perforation or perhaps both occur, and radical surgical intervention becomes inevitable. The third stage consists of second stage pathologic involvement presented for treatment either during the phase of acute obstruction or perforation or after a fistula has developed.

In the first stage the symptoms are those of acute appendicitis. There is a history of pain in the lower portion of the abdomen localized to the right side, with nausea, vomiting and some elevation of temperature. Examination reveals a well nourished person, usually with tenderness and rigidity localized to the right lower quadrant and with rebound tenderness. In the second stage the usual diagnosis from the symptoms points toward an acute intestinal obstruction associated with an inflammatory process in the right lower quadrant. There is a history of previous constipation or diarrhea and previous attacks of pain in the right lower quadrant referable to lesser degrees of partial obstruction. During the acute attack of obstruction there are obstipation, nausea, vomiting, rapid pulse, a slight rise in temperature and leukocytosis. On examination the patient usually appears to be acutely ill, and besides tenderness and rigidity in the right lower quadrant there is some degree of distention. Peristalsis may be visible abdominally, and a tumor may be palpable in the right lower quadrant. In the third stage the patient usually has a fecal or intestinal fistula as a result of, or following, previous appendectomy, with some loss of weight, general weakness and abdominal cramps. Examination usually reveals one or more abdominal scars of previous operation, with a fecal fistula.

PATHOLOGIC PICTURE

The appearance of the intestine in the early stage is that of a soggy edematous mass, which has a feeling that can be likened to that of sponge rubber. The mesentery is similarly involved. When squeezed, the intestine is almost crepitant. The edema is best visible at the

mesenteric border, where the mesentery joins the serosa of the intestinal wall. The serosa is a mottled red. The mesentery is markedly thickened, and its glands are hyperplastic. The microscopic appearance of the early lesion, as determined by biopsy (fig. 1), is that of subacute nonspecific inflammation with beginning muscular hypertrophy. From a study of the various specimens encountered, the following deductions are made: The lesion is progressive. In the earliest stages it is present in the region of the ileocecal valve, from which there is exten-

Fig. 1 (case 16).—Section of a biopsy specimen, showing subacute inflammation with fibrosis and hypertrophy of the musculature.

sion orally. Usually oral to the main hypertrophic mass, small oval ulcerations, ranging from 0.5 to 1 cm. in diameter, are present on the mesenteric surface of the ileum. In the more advanced stages ulcerations are noted throughout the entire length of the hypertrophic mass. The normal intestinal folds disappear completely. Mucosal ulceration occurs. Because of the rounding off of the sharp edges of the remaining nonulcerated mucous membrane as it converges into the surrounding

ulcerations, there is formed a hobnail appearance of the mucosa, which in its exaggerated form looks like polyposis. As the lesion involves the ileocecal valve, this becomes hyperplastic, rigid and unyielding, finally developing into a stenotic membrane. The lesion is not confined to the mucosa. In the muscularis there is an inflammatory process accompanied by hyperplasia and fibrosis. In the earlier stages of the process the wall becomes markedly thickened, so that its diameter is several times the normal, as a result not only of hyperplasia but also of edema. Thus, the transverse diameter of the wall of the intestine at this time is much greater than normal. Simultaneously, however, the lumen of the bowel becomes smaller, and in extreme forms it may become almost completely obliterated (figs. 2 to 5). Before the devel-

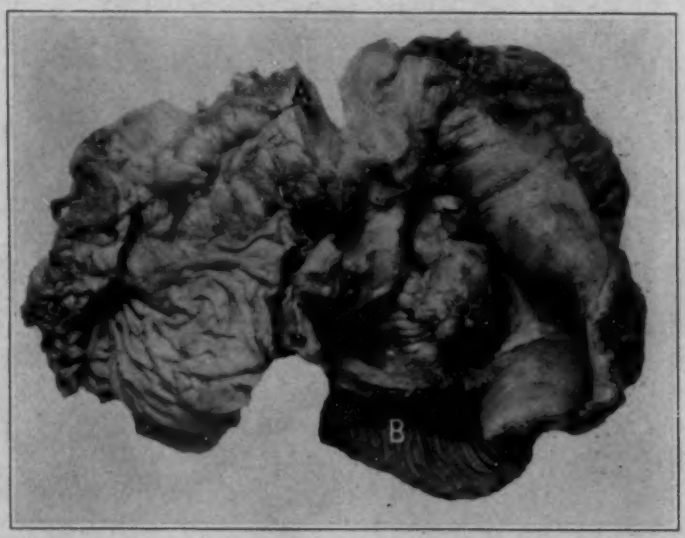

Fig. 2 (case 15).—Specimen hardened and shrunken in solution of formaldehyde, showing (*A*) tremendous thickening of the intestinal wall in cross-section at the expense of the lumen, causing clinical acute obstruction, and (*B*) the area beyond the lesion, with normal mucous membrane and lumen.

opment of the stenotic stage, that portion of the intestine which is proximal to the lesion is considerably smaller in caliber than the affected loop. When the stenotic stage develops, instead of being smaller in diameter the proximal portion of the intestine becomes larger as a result of dilatation accompanying the obstruction. As the lesion progresses, the contraction of the fibrous tissue deposited in the wall during the stage of inflammation becomes manifest. The transverse diameter of the intestine becomes less than formerly, and the whole process assumes the appearance of a rubber hose. The appearance is characteristic at operation, and even on roentgen examination no peristaltic movements

are seen in this section, whereas active peristalsis can be seen elsewhere. The mucosal ulcerations finally diminish in number, being replaced by an atrophic condition and a more marked stage of the polypoid hyperplasia previously described. Small tubercle-like structures develop on the serosal surface.

Perforation was seen in 2 cases in our series. In the first case the perforation was very extensive, involving several parts of the bowel

Fig. 3 (case 15).—Low power magnification of the cross-section at (*A*) in figure 2, showing marked encroachment of the thickened wall on the lumen. The membrane is absent in places as a result of ulceration; at other points it seems polypoid.

at one time, the ileum, the cecum and the sigmoid flexure being involved. In the second case, only the ileum was involved. In both cases the perforation was of the chronic variety. We know of no case in which a free perforation in the peritoneal cavity was encountered in the acute

stage. Apparently the tendency toward perforation is counteracted by adhesions to neighboring viscera, the omentum or the parietal peritoneum, so that when ulceration occurs through all the coats of the small intestine, the discharge of the intestinal content is either into some other organ or out on the surface, but not into the free peritoneal cavity. Occasionally the perforation occurs into a walled-off area within the peritoneal cavity and results in an abscess, which as a rule is considered to have arisen from an appendicular inflammation. When such an abscess is drained subsequently, whether the appendix is

Fig. 4 (case 15).—Section showing ulceration of the mucosa and cellular infiltration of the wall.

removed or not, it gives rise to a chronic fistula, which cannot be closed by a simple method because of the underlying inflammatory disease of the intestine.

Microscopically, the sections show various degrees of acute, subacute and chronic inflammatory change throughout the wall of the intestine, particularly in the mucosa, where there is also marked destruction of an irregular type. In some instances the entire mucosa is gone; at other

times only the surface epithelium is destroyed. There is a marked infiltration with polymorphonuclear cells, lymphocytes, plasma cells and fibroblasts. In some cases, foreign body giant cells are noted in considerable quantities, as a result of an inflammatory reaction around inclusion particles of foreign matter in the depth of the ulcers. The histologic observation of these cells plus the appearance on the serosa of small elevations discretely placed, simulating tubercles, is probably responsible for the classification of a great many of the cases in previous reports as instances of tuberculosis of the intestines.

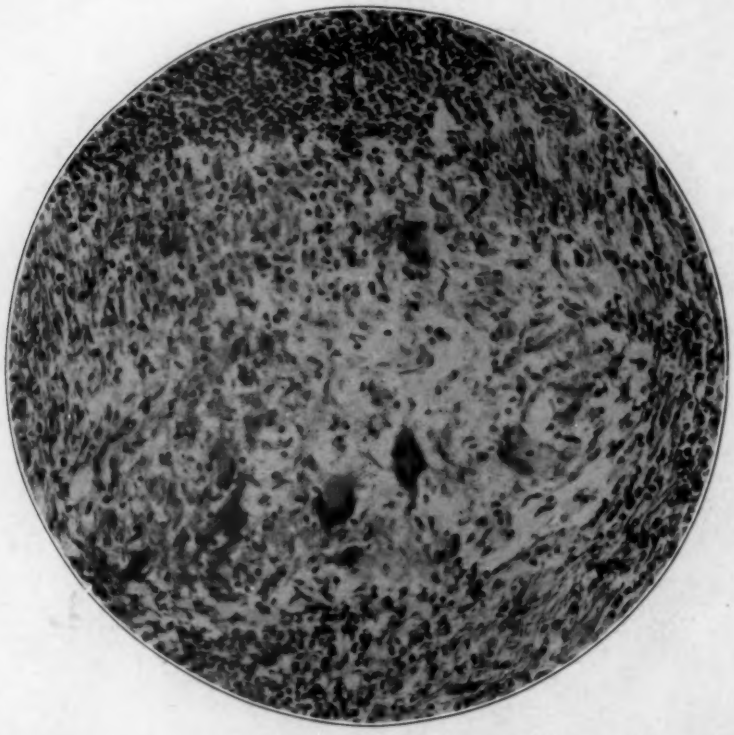

Fig. 5 (case 15).—High power magnification, showing the nodular form of cellular infiltration, with giant cells within the musculature.

ROENTGEN OBSERVATIONS

Satisfactory roentgen examination of the ileum may be accomplished in two ways. One is by giving a barium sulfate meal early in the day and observing its progress frequently from the fifth until the ninth hour. The best results are obtained by following the progress of the meal fluoroscopically, with manual manipulation under the screen, and making roentgenograms at the times best indicated by the fluoroscopic examination. The other method is the giving of a barium sulfate enema

under roentgenoscopic control until the terminal portion of the ileum is filled to the required degree by reflux through the ileocecal valve. The large intestine is then emptied by defecation, and the ileum remains filled with the contrast fluid. Normally the final loop of the ileum rises up out of the pelvis toward the ileocecal valve. In the early stages of the disease, the earliest finding in this part of the ileum is an absence of variation in the diameter of the tube, denoting a degree of rigidity which parallels the amount of cicatrization. Segmentation and peristalsis are not noted. As the process progresses, a constant filling defect in this terminal portion of the ileum develops, the extent of which depends, of course, on the degree of stenosis. Still later, when the filling defect becomes great enough, it is replaced by a thin, irregular stringy shadow caused by the very small amount of contrast medium which has passed through the stenotic opening. This appears throughout the entire extent of the filling defect, running from the beginning of the lesion to its termination. With the progress of the stenosis, as low grade obstruction develops, there is dilatation of the proximal coils and a lagging of the opaque meal in the ileum for more than nine hours. Another roentgenographic characteristic is a more or less fixed position of the loop, so that where the rigidity of the tube with lack of peristalsis is noted or where the stringy trace of barium of the stenosed ileal lesion is seen it remains more or less fixed in relatively the same position on successive plates even after several days. Kantor pointed out the roentgenologic criteria for differential diagnosis from a filled appendix: the line of the right sacro-iliac synchondrosis, normal spasm of the ileum without organic circumscribed lesions, tuberculoma, stenosing sarcoma and ileal syphilis.

REVIEW OF THE LITERATURE

Nonspecific inflammatory disease of the intestinal tract has been known for many years. It has been reported as occurring in many portions of the gastro-intestinal tract. Table 3 is a compilation of reports on 126 cases, showing the various sites of occurrence of the lesion and giving the names of the authors. Many other cases have been mentioned in various papers, but histories were not given, so that the cases were unavailable for statistical study.

Of 62 reports in which the sex was stated, the lesion occurred in males 33 times and in females 29 times. The incidence according to age was as follows: first decade, 6; second decade, 10; third decade, 12; fourth decade, 15; fifth decade, 13; sixth decade, 6, and seventh decade, 5. These 67 cases were collected from reports in which the age was stated.

In the histories of 100 of the 126 collected cases the surgical therapy was clearly reported and the ultimate results were stated. Table 4 is a

summary of the cases in which cure resulted. Table 5 is a summary of the cases in which death occurred either immediately or within the next few months after operation. In addition to the tabulated cases there were 4 in which neither cure nor death had occurred at the time the report was made. The data are given in table 6.

TABLE 3.—*Site of the Lesion in 126 Cases Reported in the Literature*

Site	Number of Cases	Author
Terminal portion of ileum...	48	Coffen (J. A. M. A. **85**: 1303 [Oct. 24] 1925); Crohn, Ginsburg and Oppenheimer;[17] Clute;[4] Rockey;[3] Harris, Bell and Brunn;[6] Colp;[8] Crohn;[9] Mock;[2] Erdmann and Burt;[14] Homans and Hass;[19] Ladd;[7] Bissell (Ann. Surg. **99**: 957, 1934); Williams (Virginia M. Monthly **60**: 728, 1934); DeCourcy;[5] Jackman;[18] Cushway (Illinois M. J. **66**: 525, 1934)
Ascending colon and cecum..	13	Moschcowitz and Wilensky;[13] Eggers;[21] Peterson;[10] Brown, Bargen and Weber;[16] Löwen (Deutsche Ztschr. f. Chir. **129**: 221, 1914); Körte (Arch. f. klin. Chir. **118**: 138. 1921)
Sigmoid flexure.....	10	Proust;[1] Moynihan (Edinburgh M. J. **21**: 228, 1907); Robson (Brit. M. J. **1**: 425, 1908); Dalziel (Brit. M. J. **2**: 1068, 1913); Monserrat (Brit. M. J. **2**: 65, 1907); Braun [12]
Cecum.....	8	Jeffries (J. M. A. South Africa **2**: 184, 1928); Mock;[2] Nemilov (Arch. f. klin. Chir. **153**: 346, 1928); Körte; Tietze (Ergebn. d. Chir. u. Orthop. **12**: 211, 1920)
Cecum and terminal portion of the ileum	9	Dalziel; Moschcowitz and Wilensky;[13] Mock;[2] Erdmann and Burt;[14] Gordon;[15] Brown, Bargen and Weber;[16] Cushway
Jejunum.....	7	Dalziel; Harris, Bell and Brunn;[6] Brown, Bargen and Weber [16]
Ascending colon, cecum and terminal portion of ileum	4	Erdmann and Burt;[14] Crohn;[9] Donchess and Warren (Arch. Path. **18**: 22 [July] 1934); Körte
Middle portion of the ileum.	3	Dalziel
Transverse colon.....	3	Moynihan; Dalziel; Robson
Ascending colon.....	3	Robson; Moschcowitz and Wilensky [13]
Stomach.....	3	Mock [2]
Entire small intestine.....	2	Dalziel
Entire lower portion of the small intestine	1	Crohn [9]
Jejunum and ileum.....	3	Ladd;[7] Corr and Boeck;[11] Schapiro (J. Mt. Sinai Hosp. **1**: 121 [Sept.] 1934)
Omentum and secondary intestinal involvement	2	Proust;[1] Mock [2]
Splenic flexure and small intestine	1	Robson
Splenic flexure and stomach	1	Mock [2]
Splenic flexure.....	1	Moschcowitz and Wilensky [13]
Transverse colon and splenic flexure	1	Moynihan
Hepatic flexure.....	1	Mock [2]
Hepatic flexure and ascending colon	1	Mock [2]
Cecum and ileocolic valve...	1	Golob (M. J. & Rec. **135**: 390, 1932)

A study of the aforementioned tables reveals the following facts: The general mortality was 14 per cent. Of the 65 cases in which resection was performed there was recurrence of the original lesion in spite of radical resection in 10, or 15 per cent. In 6 of these a second operation resulted in cure; in 3 there was a fatal outcome after a second operation, and in 1 of the 10 further surgical procedure had not been carried out at the time of the report. Simple side-tracking operations were performed in 15 cases and resulted in cure in 13 cases, in death in 1 case and in persistent external fistula in 1 case. In 8 cases the early form of the lesion was encountered, and although no intestinal

TABLE 4.—*Cases in the Literature in Which Surgical Intervention Caused a Cure*

Authors*	Case No.	Site of Lesion	Surgical Procedure
Proust [1]	1	Colon	Multiple resection
	2	Sigmoid flexure	Incision and drainage (no operation on intestine)
Moynihan	3	Transverse colon and splenic flexure	Side-tracking
Robson	4	Sigmoid flexure	Colostomy
	5	Small intestine	Ileostomy
	6	Transverse colon	Side-tracking
	7	Ascending colon	Side-tracking
	8	Splenic flexure and small intestine	Double side-tracking
Dalziel	9	Jejunum	Resection
	10, 11	Cecum and ileum (2 cases)	Type not stated but surgical procedure was carried out
	12	Sigmoid flexure	
	13	Transverse colon	
	14	Middle portion of ileum	
Coffen	15	Terminal portion of ileum	Multiple resection (2) (recurrent intestinal obstruction)
Moschcowitz and Wilensky [13]	16	Cecum and ileum	Multiple resection (3) (recurrent intestinal obstruction)
	17	Cecum and ascending colon	Resection
Jeffries	18	Cecum	Side-tracking
Mock [2]	19	Sigmoid flexure	No operation on intestine
	20	Pyloric mass	Resection
	21	Gastro-intestinal mass	Resection
	22	Splenic flexure and stone	Resection (multiple; fecal fistula)
	23	Terminal portion of ileum and cecum	Resection
	24	Ileum	Resection
Golob	25	Cecum and cecal valve	Resection
Crohn, Ginzburg and Oppenheimer [17]	26	Terminal portion of ileum	Resection (exact operation for individual case not given)
	27-38	Terminal portion of ileum	
Erdmann and Burt [14]	39	Ileocecal junction	Resection
	40	Terminal portion of ileum	Resection
	41	Cecum, ascending colon, terminal portion of ileum	Resection
Clute [4]	42	Terminal portion of ileum (mesenteric abscess)	Incision and drainage of abscess (no operation on intestine)
	43	Terminal portion of ileum	Side-tracking
Gordon [15]	44	Ileocecal mass	Resection
Eggers [21]	45	Cecum and ascending colon	Side-tracking (transection of small intestine)
	46	Terminal portion of ileum	(1) side-tracking (multiple operations); transection of small intestine (2) For obstruction by a band of adhesions
Rockey [3]	47	Terminal portion of ileum	No intestinal operation
	48		
	49		Appendectomy
	50		
Harris, Bell and Brunn [6]	51	Terminal portion of ileum	(1) Drainage for appendicitis (2) Resection of terminal portion of ileum, ascending colon and cecum
Homans and Hass [19]	52	Terminal portion of ileum	Resection
Ladd [7]	53	Lower portion of jejunum	Multiple resection
		Terminal portion of ileum	(1) Exploratory laparotomy and biopsy (2) Resection (3) Resection
	54	Terminal portion of ileum	Resection
Bissell	55	Terminal portion of ileum	Multiple operations (1) Resection (Mickulicz) (2) For intestinal obstruction (bands)
	56	Terminal portion of ileum	Resection
Colp [8]	57	Terminal portion of ileum	(1) Appendectomy; (2) resection
Williams	58	Small intestine	Resection

TABLE 4.—*Cases in the Literature in Which Surgical Intervention Caused a Cure —Continued*

Authors*	Case No.	Site of Lesion	Surgical Procedure
Crohn [9]	59	Terminal portion of ileum Ascending colon Hepatic flexure Sigmoid	(1) Appendectomy followed by progression of symptoms Multiple resections
	60	Terminal portion of ileum	Resection
	61	Terminal portion of ileum	Resection
Brown, Bargen and Weber [16]	62 to 68	Ileum	Side-tracking (3); resection (4) (1 or 2 stages)
	69	Jejunum	Side-tracking
	70 to 75	.....	Side-tracking (1); resection (5) (1 or 2 stages)
DeCourcy [5]	76	Terminal portion of ileum	No intestinal operation (appendectomy)
Donchess and Warren	77	Cecum Ascending colon Terminal portion of ileum	Resection
Jackman [18]	78	Terminal portion of ileum	Resection
	79	Terminal portion of ileum	Resection
Schapiro	80	Terminal portion of jejunum and upper portion of ileum	Resection
Cushway	81	Terminal portion of ileum and cecum	Resection
	82	Terminal portion of ileum	Resection

* Reference to authors not mentioned in the text will be found in table 3.

procedure was instituted, cure resulted (Proust;[1] Mock;[2] Rockey;[3] Clute;[4] DeCourcy[5]). In 6 other cases in which the early stage was observed at the primary operation and no intestinal procedure was instituted, there was marked progression of the intestinal disease, necessitating further operation. In this group subsequent resection resulted in cures in 4 cases, death in 1 case and intractable fistula in 1 case (Harris, Bell and Brunn;[6] Ladd;[7] Colp;[8] Crohn;[9] Peterson;[10] Corr and Boeck[11]).

1. Proust, M. R.: Tumeur para-intestinale [Rapport par Lejars], Bull. et mém. Soc. de chir. de Paris **33**:1158, 1907.

2. Mock, H. E.: Infective Granuloma, Surg., Gynec. & Obst. **52**:672 (March) 1931.

3. Rockey, E. W.: Thickening of Terminal Ileum with Mesenteric Adenitis in Children, Northwest Med. **32**:145 (April) 1933.

4. Clute, H. M.: Regional Ileitis, S. Clin. North America **13**:561 (June) 1933.

5. DeCourcy, J. L.: Terminal Ileitis Simulating Acute Appendicitis (Case Report), J. Med. **15**:216 (June) 1934.

6. Harris, F. I.; Bell, G. H., and Brunn, H.: Chronic Cicatrizing Enteritis: Regional Ileitis (Crohn), Surg., Gynec. & Obst. **57**:637 (Nov.) 1933.

7. Ladd, William, in discussion on Homans and Hass.[19]

8. Colp, R.: Case of Nonspecific Granuloma of Terminal Ileum and Cecum, S. Clin. North America **14**:443 (April) 1934.

9. Crohn, B. B.: Broadening Conception of Regional Ileitis, Am. J. Digest. Dis. & Nutrition **1**:97 (April) 1934.

10. Peterson, E.: Non-Specific Granuloma of Ileum, Ann. Surg. **97**:130 (Jan.) 1933.

11. Corr, P., and Boeck, W. C.: Chronic Ulcerative Enteritis (Regional Ileitis), Am. J. Digest. Dis. & Nutrition **1**:161 (May) 1934.

Fecal fistula occurred in 9 cases (Braun;[12] Moschcowitz and Wilensky;[13] Mock;[2] Erdmann and Burt;[14] Gordon;[15] Crohn;[9] Corr and Boeck;[11] Brown, Bargen and Weber[16]) exclusive of the cases observed in Mount Sinai Hospital, reported on by Crohn, Ginzburg and Oppenheimer,[17] who did not mention the percentage of occurrence. A review of the fatal cases revealed the following: In 3 the condition was recurrent after previous resection; in 4 the jejunum was extensively involved, and in 5 there was extensive intestinal involvement, the entire intestine being involved in 2, the entire lower portion of the small intestine in 1 and extensive portions of the jejunum in 2. We may predicate from

TABLE 5.—*Cases in the Literature in Which Death Followed Surgical Procedure* †

Authors*	Case No.	Site	Procedure
Braun [12]	1	Sigmoid flexure	Multiple resection (recurrent)
Dalziel	2	Whole intestine	No operation on intestine
	3	Whole intestine	No operation on intestine
Moschcowitz and Wilensky [13]	4	Ascending colon and small intestine	Multiple resection (recurrent); resection
Mock [2]	5	Stomach	Resection
Erdmann and Burt	6	Terminal portion of ileum Ascending colon	Multiple resection (recurrent) Resection (terminal portion of ileum and ascending colon)
Harris, Bell and Brunn [6]	7	Terminal portion of ileum	Resection
	8	Jejunum	Resection
Crohn [9]	9	Whole lower portion of intestine	Resection
Corr and Boeck [11]	10	Terminal portion of ileum and jejunum (extensive involvement)	Side-tracking (at previous operation of app.) bowel lesion not advanced at time of side-tracking; extensive involvement
Brown, Bargen and Weber [16]	11	Jejunum	Procedure not stated but no resection done
	12	Jejunum (extensive involvement)	Closure of fistula
	13	Ileum	
Cushway	14	Terminal portion of ileum Cecum	Laparotomy

* Reference to authors not mentioned in the text will be found in table 3.

† Factors influencing mortality were: recurrence after previous operation (3 cases); jejunal involvement (4 cases), and extensive involvement (5 cases).

12. Braun, H.: Ueber entzündliche Geschwülste am Darm, Deutsche Ztschr. f. Chir. **100**:1, 1909.

13. Moschcowitz, Eli, and Wilensky, A. O.: Nonspecific Granulomata of Intestine, Am. J. M. Sc. **166**:48 (July) 1923.

14. Erdmann, J. F., and Burt, C. V.: Non-Specific Granuloma of Gastro-Intestinal Tract, Surg., Gynec. & Obst. **57**:71 (July) 1933.

15. Gordon, Donald: Non-Specific Granuloma of Ileum, Ann. Surg. **97**:130 (Jan.) 1933.

16. Brown, P. W.; Bargen, J. A., and Weber, H. M.: Regional Enteritis, Proc. Staff Meet., Mayo Clin. **9**:331 (May 30) 1934.

17. Crohn, B. B.; Ginsburg, L., and Oppenheimer, G.: Regional Ileitis, J. A. M. A. **99**:1323 (Oct. 15) 1932.

this that extensive involvement, recurrence of the lesion and jejunal involvement are unfavorable factors as far as prognosis as to life is concerned.

A review of the reports of cases in which cure occurred revealed 54 instances of radical resection and 13 instances of side-tracking operations. There were 8 cases in which an early lesion was encountered in which surgical procedure was not performed on the intestine.

Following the publication of the paper of Crohn, Ginsburg and Oppenheimer [17] in 1932, a fair number of isolated cases, in groups of 1, 2 or 3, were reported in which the lesion in the terminal portion of the ileum was described and the condition was considered a definite disease entity (Crohn, Ginzburg and Oppenheimer; [12] Clute; [4] Jackman; [18] Colp [8]). However, other papers soon appeared in which the authors expressed the opinion that the disease in the terminal portion of the ileum was not especially different from similar inflammatory involvement of other portions of the intestine (Homans and Hass; [19]

TABLE 6.—*Cases in the Literature with Improvement and Persistent Fistulas or Recurrence (with No Further Therapy at the Time the Case Was Reported)*

Author	Case No.	Site	Procedure	Result
Peterson [10]	1	Cecum, ascending colon	Side-tracking	Persistent fistula
Homans and Hass [19]	2	Terminal portion of ileum	Resection	Recurrent
	3	Terminal portion of ileum	Resection	Improvement
Ladd [7]	4	Lower portion of jejunum and terminal portion of ileum	Multiple resections	Improvement

Harris, Bell and Brunn; [6] Crohn [9]). The similarity of the lesion as it is described by various pathologists, regardless of the portion of the intestine involved, and the clinical course in the cases cited in the literature in which the pathologic process was not limited to the terminal portion of the ileum make us believe that the location in the terminal portion is merely more frequent than elsewhere but that the underlying disease is the same if regarded from the point of view of the pathologist. Clinically, the syndromes encountered are different according to the location of the pathologic process. Thus, the early stage of ileitis of the terminal portion gives the picture of irritation in the right lower quadrant of the abdomen associated with varying amounts of obstruction of the small intestine or of varying degrees of

18. Jackman, W. A.: Localized Hypertrophic Enteritis as Cause of Intestinal Obstruction, Brit. J. Surg. **22**:27 (July) 1934.

19. Homans, J., and Hass, G. M.: Regional Ileitis: Clinical Not Pathological Entity, New England J. Med. **209**:1315 (Dec. 28) 1934.

severity of diarrhea, depending on the stage of the disease. Involvement of the sigmoid flexure presents the picture characteristic of inflammatory or obstructive disease of the large intestine. A lesion of the jejunum or the upper portion of the ileum gives the picture of inflammatory or obstructive disease of the small intestine. It is possible that the greater frequency of ileal involvement may be due to greater susceptibility of this portion of the intestinal tract to infection, as is noted particularly in children by the development of mesenteric adenitis to a greater extent in the mesentery draining the terminal portion of the ileum than elsewhere in the intestinal tract.

Etiology.—For a theoretical discussion on etiology reference should be made to the papers of Mock [2] and Ginzburg and Oppenheimer.[20] Because of the cicatrizing tendencies and the possible similarity to cicatrizing tendencies found in the rectum in cases of lymphogranuloma inguinale, we carried out the Frei test, with negative results, in 4 cases in which resection was performed and also in 2 cases in which resection was not done.

DIAGNOSIS

We should like to emphasize again the steps that must be taken to make the diagnosis. To begin with, any case in which there are symptoms of inflammatory intra-abdominal disease with peritoneal irritation as well as symptoms of varying degrees of intestinal obstruction must be considered as possibly an instance of regional ileitis. Long continued duration of symptoms is characteristic in many of these cases, although occasionally the clinical course is short. We stress the importance of the roentgen findings, particularly as regards the use of the barium enema. The barium meal may also be used, but to less advantage when the lesion is in the terminal portion of the ileum. It will be of much greater importance if the lesion is in the upper portion of the intestine. Serial plates must be studied to establish the diagnosis. General constitutional symptoms characteristic of inflammation of the intestinal tract, such as fever, leukocytosis, increase in the pulse rate, etc., while present, offer little differential diagnostic aid.

TREATMENT

A review of the collected literature as well as of our own cases establishes certain guides for therapy:

The condition may be subdivided into three types: (*a*) the early lesion, (*b*) the moderately advanced but not obstructive lesion and (*c*) the advanced stenotic or fistulous process.

20. Ginzburg, L., and Oppenheimer, G. D.: Non-Specific Granulomata of the Intestines; Inflammatory Tumors and Strictures of the Bowel, Ann. Surg. **98:** 1046 (Dec.) 1933.

The early lesion in many instances is spontaneously cured (in 6 cases in our series and in 8 in the literature). That there are many cases in which spontaneous cure has occurred without diagnosis or operation seems probable. Therefore, conservatism in the management of the early lesion is indicated. The therapeutic value of roentgen ray treatment is a moot question, the number of cases in which it was given being too small to allow of conclusions (Eggers,[21] 2 cases after side-tracking; Peterson,[10] 1 case, and ourselves, 4 cases). On the other hand, in 6 cases reported in the literature there was subsequent marked progression, necessitating radical surgical procedure. Also, in 13 cases of advanced lesion a side-tracking operation resulted in cure. This suggests the advisability of side-tracking in cases in which the process has advanced beyond the very early stage. (In this connection, it must be remembered that the personal equation is an important factor in grading the stage of the lesion, since individual experience has not been very extensive.)

With the moderately advanced lesion, the choice of procedure lies between a side-tracking operation and resection. The desirability of eradicating a focus in which at this stage the chance for regression to normal is probably very slight favors resection as the procedure of choice. If side-tracking is decided on, occlusion of the ileum proximal to the lesion is advisable.

In cases of the advanced stage resection is indicated. Whether it is performed in one or more stages depends on many factors. If the condition is complicated by acute obstruction, one stage resection is to be avoided. If the obstruction is of long standing, an enterostomy is the best immediate procedure. This is subsequently followed by resection. If the patient's condition warrants, the obstruction may be relieved by a side-tracking anastomosis. Here, however, delay in proceeding with the resection is fraught with the danger of rapid progression of the lesion, necrosis and suppuration in the mesentery (Braun,[13] case 2) and possibly peritonitis from the perforation of such an abscess or even of the intestine itself. The experiments on dogs with acute obstruction and the clinical experience of Holm [22] emphasize this point.

With complicating fistulas the resection becomes more difficult. The judgment of the operator and the extent of the lesion must determine the amount of intestine to be resected. In our 2 cases only the small

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21. Eggers, Carl: Non-Specific Granuloma of Ileum, Ann. Surg. **97**:130 (Jan.) 1933.

22. Holm, C. E.: Fate of the Sidetracked Loop of Ileum Following Lateral Anastomosis for Complete, Benign Obstruction, Surg., Gynec. & Obst. **56**:746 (April) 1933.

intestine was resected, the openings into the sigmoid flexure and cecum being closed by purse-string sutures. Both operations were carried out in one stage. Here also it may be advantageous to divide the procedure into two stages by a preliminary side-tracking operation.

SUMMARY

We have presented an analysis of our 17 cases of nonspecific inflammatory disease of the ileum and have analyzed the condition in 100 cases reported by others. From this study we have made generalizations regarding diagnosis, prognosis and therapy.

EFFECT OF INFLAMMATION ON THE EPIPHYSES

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The known and commonly described infections which occur in human beings at the epiphysis or at its union with the diaphysis are as follows:

A. Acute osteomyelitis.

B. Chronic involvement; associated with tuberculosis, syphilis, undulant fever (rare) or actinomycosis (rare).

C. Gradual necrosing lesions of bone the etiology of which is disputed:

1. Köhler's disease of the tarsal navicular bone; 2. Freiberg's disease of the heads of the metatarsal bones; 3. Kienböck's disease of the carpal semilunar bone and many other bones; 4. Koenig's osteochondritis dissecans of the medial condyle of the femur; 5. apophysitis of the os calcis (Sever); 6. aseptic necrosis of the head of the femur, the radius, the humerus or the distal epiphysis of the femur; 7. Osgood-Schlatter's disease of the tibial tubercle; 8. Legg's or Calvé-Perthes' disease, osteochondritis juvenilis.

In this paper it would be impossible to deal with this great variety of diseases about which so much has been contributed by clinicians, surgeons, roentgenologists and research workers of this and other countries; so I am limiting myself to the subject of acute osteomyelitis.

The interest that has been shown in the recent adjuncts to the treatment of osteomyelitis (Orr's [1] method and Dolman's staphylococcus serum [2] during the acute stage and the various procedures for balancing the length of shortened extremities (Putti's,[3] Abbott's [4] and

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From the Division of Orthopedic Surgery of the Department of Surgery of the University of Western Ontario.

Read before the Section on Surgery at the Eighty-Sixth Annual Session of the American Medical Association, Atlantic City, N. J., June 13, 1935.

1. Orr, H. W.: Treatment of Osteomyelitis by Drainage and Rest, J. Bone & Joint Surg. **9**:733, 1927.

2. Dolman, C. E.: Staphylococcus Antitoxic Serum in the Treatment of Acute Staphylococcal Infections and Toxemias, Canad. M. A. J. **30**:601 (June) 1934.

3. Putti, V.: Operative Lengthening of the Femur, Surg., Gynec. & Obst. **58**: 318 (Feb.) 1934.

4. Abbott, L. C.: The Operative Lengthening of the Tibia and Fibula, J. Bone & Joint Surg. **9**:128 (Jan.) 1927.

Phemister's [5] method) during the later stages of deformity has to some extent obscured the necessity for early diagnosis and immediate adequate surgical treatment as advocated by Starr.[6]

The primary pathologic change of acute osteomyelitis appears at the diaphyseal side of the epiphyseal cartilage, probably as an infective embolus, and produces a septic infarct the same as would occur in any other tissue.

The lodging of an embolus or the collection of a group of organisms in this particular location has been accounted for anatomically [7] by the arrangement of the blood vessels and also by the fact that the direct supply of blood at the site consists of terminal arteries (fig. 1).

A clinical picture is produced which nearly always is acute and characteristic. The patient, more often a boy from 9 to 14 years of age, complains of acute pain near the end of one of his long bones, frequently

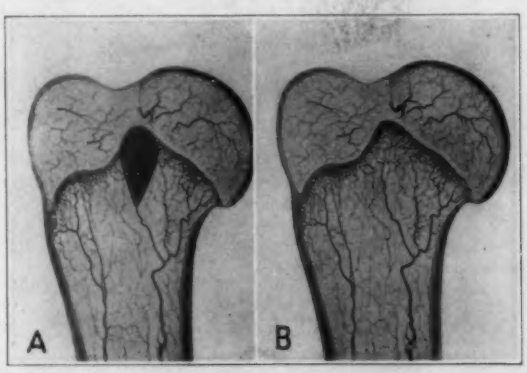

Fig. 1.—*A* shows the location of the early lesion of acute osteomyelitis in relation to the supply of blood of the epiphyseal line. *B* shows the vascular supply of bone (after Harris).

the lower end of the femur or the upper end of the tibia. A history of trauma is sometimes present, and often a scrutiny of the affected extremity reveals the site of a recently infected blister, callus or cutaneous wound or the presence of some recent cutaneous infection, or the history of a recent cold or minor ailment may be obtained. The extremity is held in a position of flexion, and although swelling is not an early sign it soon appears and is best noted by a comparison with the opposite extremity (fig. 2).

5. Phemister, D. B.: Operative Arrestment of the Longitudinal Growth of Bone in the Treatment of Deformities, J. Bone & Joint Surg. **15**:1 (Jan.) 1933.

6. Starr, C. L.: Acute Hematogenous Osteomyelitis, Arch. Surg. **4**:567 (May) 1922.

7. Harris, H. A.: The Vascular Supply of Bone with Special Reference to the Epiphyseal Cartilage, J. Anat. & Physiol. **64**:1, 1930.

Starr [8] stressed the presence of a finger point of exquisite tenderness corresponding to the site of the epiphyseal line, and I have always found it a most reliable sign. It is found by gentle testing in a line commencing above and working down toward the joint on the surface of the skin over the side of the extremity nearest the pathologic condition. No doubt exists about the exquisite tenderness of this point when found, and its presence is a great aid in making an early diagnosis.

In addition to these local findings there are usually fever, with a temperature of from 102 to 103 F. or more, and increase of the pulse

Fig. 2.—Acute osteomyelitis of three days' duration affecting the upper end of the right tibia of a 14 year old boy. Note the swelling of the limb.

rate to 120 or 140. The leukocyte count is definitely increased, the number varying in my series from 12,000 to 55,000 per cubic millimeter.

In this series I found that in several cases the condition had been diagnosed rheumatic fever and treated as such even after multiple foci had developed. The early swelling of an acute rheumatic joint and the fact that more than one joint is usually involved in rheumatic fever should aid in distinguishing this condition from acute osteomyelitis.

8. Starr, Clarence L.: Acute Infections of Bone, in The Robert Jones Birthday Volume, New York, Oxford University Press, 1928, p. 183.

However, there is an increase of synovial fluid in acute osteomyelitis when the lesion has developed, but at this stage the infection is definitely in the bone and is not articular.

In a number of my cases in which the inflammation involved the upper end of the femur, humerus, scapula, pelvis and tarsal bones,

Fig. 3.—*A*, an early stage of osteomyelitis of the lower end of the femur. Roentgenograms showed no osseous changes. The high riding patella suggests an increase of synovial fluid in the knee joint. *B*, lateral view of the bone. Note the perforation of the cortex just above the epiphyseal line posteriorly. *C*, roentgenogram of the bone twelve days later, showing the misplaced drill holes and the inflammatory process progressing just above the epiphyseal line. *D*, the same bone, showing a section of cortex removed but not near enough to the epiphysis to allow free drainage. There is more destruction of the lower end of the diaphysis than is seen in *A*.

the diagnosis of cellulitis had been made; however, this was made after the infection had existed for several days and perforation of the cortical bone had taken place. The presence of cellulitis draws attention to the fact that this condition was a complication and not the cause of the original infection. Primary cellulitis may be distinguished by the early swelling and redness which it causes and also by the fact that the pain and toxemia are not so marked as in acute osteomyelitis.

Acute septic arthritis, which like cellulitis may be a later complication of acute osteomyelitis, must also be distinguished when it occurs as a primary condition. The mono-articular character of the lesion is a troublesome diagnostic point, but the inflammatory demonstration is

Fig. 4.—*A* shows the femur of a rabbit which died eighteen hours after being inoculated with a broth culture of Staph. aureus from the patient whose femur is shown in figure 3. The lower epiphysis was separated. *B* shows the epiphysis of the rabbit separated by the acute inflammatory process.

limited to the articulation, and aspiration of the joint will settle any question of doubt which may arise.

The history of trauma may cause some doubt as to the diagnosis of the lesion, but the examination and the presence of fever, toxemia and leukocytosis will reveal the true nature of the lesion.

I have produced acute osteomyelitis in the long bones of young rabbits by injection into the blood vessels [9] of 0.5 cc. of a broth culture of

9. Kistler, Gene: Sequence of Experimental Infarction of the Femur in Rabbits, Arch. Surg. **29**:589 (Oct.) 1934.

Staphylococcus aureus, obtained from patients in the early stages of acute osteomyelitis, and I have recovered the organism again, uncontaminated, from the epiphyseodiaphyseal joint of the infected bone. The macroscopic and microscopic examination of these specimens shows the type of lesion which occurs in acute osteomyelitis caused by Staph. aureus and has a definite bearing on the type of surgical treatment that should be instituted (fig. 3).

The question of the type of operation is one of great importance. All methods consist of exposure of the infected bone. The site of the epiphyseal line should be located. The fluoroscope is of aid in this, and the approximate location of the line can be marked on the surface of the skin. The incision should extend to this line, and the epiphyseal line should be identified. Due regard should be paid to the integrity of the capsule of neighboring joints, and a consideration of the attachment of tendons and ligaments will serve as a guide to the exposure of the

Fig. 5.—Pure growth of Staph. aureus on blood agar plates. *A* is from the patient whose tibia is shown in figure 2; *B*, from the rabbit.

epiphysis. The periosteum should be incised in a linear direction to the epiphyseal line. Then the treatment of the bone itself is to be considered.

In early cases the location of the pathologic condition is best reached by the drilling of obliquely placed holes through the cortex of the bone to the epiphysis (fig. 8).

Starr [6] advised simple periosteal incision in cases in which pus is found under the periosteum. If no pus is found, the bone should be drilled or trephined obliquely to the epiphyseal line. This procedure has many advantages; it is a fairly conservative procedure compared to the removal of a large segment of the cortex or to guttering the bone or diaphysectomy, and it allows drainage. Also bacteriologic studies can be made of the fluid and débris which come from the drilled holes.

Young patients suffering from the toxemia of acute osteomyelitis do not withstand severe operations, and a comparison of the mortality among a series of patients treated by extensive guttering as compared

Fig. 6.—Photomicrograph (low power) showing experimentally produced osteomyelitis at the lower end of the femur in an 8 week old rabbit.

Fig. 7.—Photomicrograph (high power) showing the junction of the diaphysis and epiphysis. Note the acute inflammatory reaction on the diaphyseal side of the epiphyseal line.

to a series treated primarily by the more conservative drilling favors the conservative treatment.[10]

The progress in a case of acute osteomyelitis is demonstrated in figures 5, 6, 7 and 8*A*, showing various changes which occur in the bone when the region of the end of the diaphysis is not adequately drained and emphasizing the necessity of draining the bone close to the epiphyseal line. The integrity of the epiphysis is a remarkable feature and supports the idea of its having a different blood supply than the diaphysis.[7] The fundamental principle of treatment is to give early and adequate drainage as close to the site of inflammation as possible, without injuring the growing end of the diaphysis more than necessary. This calls for carefully planned incision, exposure of the bone which shows

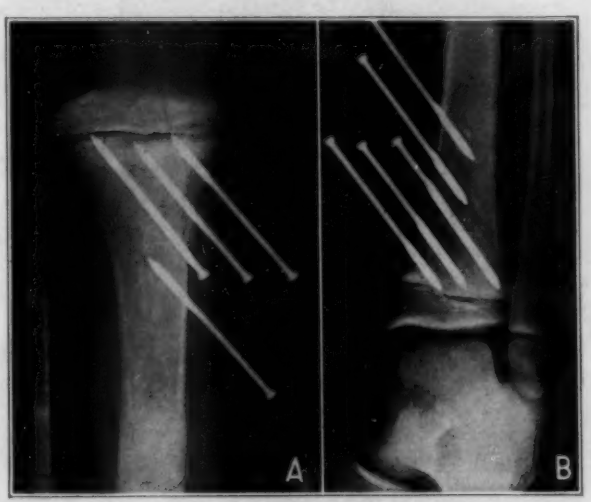

Fig. 8.—*A* shows drill holes placed obliquely to the epiphyseal line at the upper end of the tibia of a 7 year old boy. Two inch brads have been placed in the holes to afford contrast; *B* shows obliquely placed drill holes at the lower end of the tibia of the same patient. The holes would drain the cancellous bone at the end of the diaphysis, the site of the early pathologic condition of acute osteomyelitis.

the location of the epiphyseal line and careful opening of the periosteum and cortex of the bone without splintering or tearing the bone either toward the epiphysis or in the shaft. The drill fulfils these obligations and is always used by me in primary treatment of patients in whom the disease is in the early stage and those who are acutely ill. All cases are not diagnosed early, and many patients have reported to me when the suppurating process had become chronic and when the immediate indica-

10. Pyrah, L. N., and Pain, A. B.: Acute Infective Osteomyelitis: A Review of Two Hundred and Sixty-Two Cases, Brit. J. Surg. **20**:590 (April) 1933.

tion was for adequate drainage. This, I think, accounts for the high percentage of patients showing epiphyseal involvement and interference with growth, but on the other hand my conservative type of operation on the acutely ill patients and on those whose disease was in the early stages accounts for the low mortality.[11]

Pyrah and Pain [10] reported 52 deaths in 176 cases of acute osteomyelitis in which treatment was primarily by guttering, while I have treated 73 patients with involvement of 105 bones, with only 1 death by using more conservative treatment (table 2). Pyrah and Pain ended

Fig. 9.—A marked bowleg deformity in a patient who had acute osteomyelitis with metastases due to destruction of the medial part of the epiphyseal line of the upper end of the tibia and unequal growth and unsupported weight bearing.

their discussion by pointing out the necessity for a more conservative type of operation early in the disease.

A study of this series of cases of acute osteomyelitis and a comparison with the results of various methods of treatment reported in the literature shows that acute osteomyelitis is a very grave disease. The analysis of 262 cases with 71 deaths reported by Pyrah and Pain [10] in England showed a mortality of 27.1 per cent. In addition, a great many

11. Ramsay, G. A.: Personal communication.

patients go on for long periods of time with chronic discharging sinuses and acquire various degrees of lardaceous disease, and many are left with permanently damaged epiphyseal cartilage, which causes unequal length of extremities and various angular deformities such as bowleg and knock knee (figs. 8 *B*, 9 and 10).

Phemister [5] drew attention to the effect of injury and inflammation on the epiphysis and to the various deformities which may result. He advised operative arrest of the epiphyseal growth to balance the length of unequal extremities and to cure angular deformities at joints caused by unequal growth.

Fig. 10.—A marked knock knee deformity in a patient with a late stage of osteomyelitis of the upper end of the tibia, due to destruction of the lateral portion of the epiphyseal line and unequal growth.

I became interested in this type of deformity in osteomyelitis and have analyzed this series of cases to ascertain in what percentage the epiphyseal growth is interfered with by the inflammatory process (tables 1 and 2).

I found many instances of shortening of the femur and the tibia, one case of osteomyelitis of the upper end of the femur having caused shortening of the leg of 4½ inches (11.4 cm.) and one of the upper end of the tibia having caused shortening of 2½ inches (6.3 cm.).

Bowleg and knock knee caused by arrest of either the inner or the outer half of the epiphyseal line of the lower end of the femur or of the upper end of the tibia occurred several times. Remarkable improvement

TABLE 1.—*A Statistical Study of Acute Hematogenous Osteomyelitis Treated by the Surgical Service of the University of Western Ontario (1926-1935)*

Bones Involved	No. of Cases	Average Age	Sex	Side	End*	Metastatic Involvement	Arrest of Growth with Epiphyseal Involvement	No Arrest; Epiphysis Intact	Not Traced	Too Recent	Died
Femur	38	11.5	M 27 F 11	R 20 L 28	U 10 L 28	8	18	16	3	1	1
Tibia	24	11	M 22 F 8	R 20 L 14	U 18 L 12	4	15	14	3	2	0
Humerus	10	9	M 3 F 4	R 6 L 4	U 3 L 7	3	5	2	2	1	0
Pelvis	6	9	M 3 F 3	R 3 L 3	.....	2	3	2	0	1	0
Ulna	5	10	M 3 F 2	R 4 L 1	U 0 L 5	5	3	2	0	0	0
Os calcis	4	10	M 2 F 2	R 1 L 3	.....	3	4	0	0	0	0
Radius	2	7.5	M 1 F 1	R 2	..... L 2	1	1	1	0	0	0
Scapula	2	5	M 1 F 1	R 1 L 1	.....	2	1	0	0	1	0
Clavicle	1	12	F 1	R 1	.....	1	0	1	0	0	0
Fibula	1	16	M 1	R 1	L 1	1	0	1	0	0	0
Sternum	1	19	M 1	Body	.....	1	0	1	0	0	0
Skull	1	11	M 1	Frontal	.....	1	0	1	0	0	0
Totals	105	9.5	M 65 F 33	R 58 L 45	U 31 L 44	32	50	41	8	6	1

* U indicates upper; L, lower.

TABLE 2.—*Analysis of the Cases Listed in Table 1*

Total number of patients	73
Total number of bones involved	105
Number of bones showing interference with growth	50*
Number of bones showing no interference with growth	41
Number of cases in which patient was not traced	8
Number of cases too recent for opinion	6
Number of deaths	1
Percentage of bones showing involvement of growth	55.5

* Two bones showed longitudinal overgrowth.

has occurred in some of these cases by the patient's wearing Thomas walking calipers and relieving the site of deformity of the strain of bearing weight and using a strap.

In the case of the upper end of the femur and lower end of the humerus, involvement of the joint was the rule, owing to the anatomic relation of the epiphyseal line to the joint, and in these cases the epiphysis was usually destroyed.

The penalty for delayed diagnosis is the prolongation of toxemia, the multiplication of local complications, the development of secondary lesions in other bones and the destruction of the growing cells in the epiphyseal line, which causes various deformities, all of which prolong the course of the disease and increase the mortality.

TABLE 3.—*The Effect of Inflammation on the Epiphysis, Indicating the Location and Degree of the Arrest in Growth*

Location	Complete	Incomplete
Femur*	Lower 2 Upper 5	Lower 8 Upper 5
Tibia	Lower 0 Upper 2	Lower 4 Upper 9
Humerus	Lower 3 Upper 0	Lower 2 Upper 0
Os calcis	3	1
Ulna	..	Lower 3
Pelvis	..	3
Scapula	..	1
Radius	..	Lower 1
Total	15 (16.2%)	35 (38.8%)

* Two bones showed longitudinal overgrowth.

SUMMARY

A series is reported of 73 consecutive cases of acute hematogenous osteomyelitis, affecting 105 bones, with 1 death, which occurred on the third day after admission of an 8 year old girl with the disease affecting the lower end of the left femur.

The pathologic characteristics, the symptomatology and the operative treatment are briefly discussed.

An analysis of the series is made, especially in regard to the effect of inflammation on the epiphyses, with complete or incomplete interruption of growth, which occurred in 50 bones, or 55.5 per cent.

In two cases in which the condition affected the lower end of the femur there was longitudinal overgrowth of 2½ inches (6.3 cm.) and 1 inch (2.5 cm.), respectively.

ABSTRACT OF DISCUSSION

DR. F. C. KIDNER, Detroit: Dr. Johnston's paper is very much to the point in that it accents the necessity of early diagnosis and conservative operation

performed at the proper point in the affected bone. His insistence on the classic principles is most worth while. If he approaches too close to the diaphyseal line of the epiphysis with the drill holes, perhaps it is possible to explain part of his large incidence of arrest in growth by damage to the diaphyseal cartilaginous cells in the same way that Dr. Compere explained arrest of growth following fractures. My experience with failure in growth following osteomyelitis has been more extensive than his. In the cases of failure in growth Dr. Johnston did not distinguish between those in which he operated himself and those in which less well informed surgeons operated.

Dr. J. Dewey Bisgard, Omaha: Dr. Johnston should be congratulated on the low mortality rate that he has had in cases of acute osteomyelitis. His unusually high incidence of disturbance in growth, I believe, can be explained by the fact that he carries the operation very close to the epiphyseal cartilage and into the juxtocartilaginous portion of the diaphysis, where longitudinal growth takes place. However, the deformities resulting from disturbance in growth constitute rather a small price to pay for the very low mortality rate that he has had in his series of cases.

Dr. R. A. Y. Johnston, London, Canada: Dr. Kidner mentioned that there is no doubt that those drill holes placed too close to the diaphyseal line of the epiphysis arrested growth. I wished to emphasize the necessity for placing them there. In practical cases, one does not try to go more than to the second or third drill hole or very far into the diaphyseal end of the bone. The incidence of deformity in the series I presented and comparison of the mortality in this series with that in a series of 262 cases in England that were reported in the *British Journal of Surgery* in April 1933, wherein 176 children with acute osteomyelitis were treated by the primary guttering operation and 52 per cent died, indicate, I believe, the practicality of my procedure. I allow supported weight bearing when the pathologic process has become quiescent, and as Dr. Ramsay, the director of our clinic, was trained by the Liverpool School, we sometimes persist with supported weight bearing. A study of the percentage of arrests of growth in the femur and the tibia in this series would indicate that continued protection does not protect against epiphyseal deformity.

THE PUBIC BONES AND THEIR SYMPHYSIS

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The roentgenographic configurations cast by the pubic bones and their symphysis have aroused my special interest. In an effort to interpret these more fully, studies have been made on seventy-five specimens of this anatomic region,[1] obtained post mortem. The specimens were examined histologically and roentgenographically for the purpose of elucidating the following questions: 1. How does the symphysis pubis develop and is it a true joint? 2. What should be considered its normal appearance until puberty? 3. Are there any growth changes after adolescence 4. What changes in this area after middle age may be deemed normal? 5. What changes occur in the symphysis pubis during pregnancy? 6. What modifications are produced in this region by the local presence of pathologic tissue?

METHOD OF STUDY

In the course of seventy-five autopsies, the portion of the body in the region of the symphysis pubis was removed. The specimens came from persons whose ages at the time of death ranged from less than 1 year to 75 years (table). Specimens from a premature and from a newly born infant were also examined. On removal, each specimen was roentgenographed in its anterior-posterior plane, after which it was cut serially in the same plane into several thin portions. Each

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From the Laboratory Division, Hospital for Joint Diseases.

* Brown Orthopedic Research Fellow.

1. Hunter, W.: Remarks on the Symphysis of the Ossa Pubis, Med. Obs. Soc. Phys. London **2**:321, 1762. Zaglas: On the Symphysis Pubis and Its Contained Cavity, Month. J. M. Sc. **13**:489, 1851. Aeby, C.: Ueber die Symphysis Ossium Pubis des Menschen nebst Beiträgen zur Lehre vom hyalinen Knorpel und seiner Verknöcherung, Ztschr. f. rat. Med. **4**:1, 1858. Fick, R.: Bemerkungen über die Höhlenbildung am Schemfugenknorpel, Anat. Anz. **19**:307, 1901. Zulauf, C.: Die Höhlenbildung am Symphysenknorpel, Arch. f. Anat. u. Physiol. **1**:95, 1901. Loescheske, H.: Untersuchungen über Entstehung und Bedeutung der Spaltbildungen in der Symphyse, sowie über physiologische Erweiterungsvorgänge am Becken Schwangerer und Gebärender, Arch. f. Gynäk. **96**:525, 1912. Putschar, W.: Zur Pathologie der Symphyse, Verhandl. d. deutsch. path. Gesellsch. **25**:214, 1930; Entwicklung, Wachstum und Pathologie der Beckenverbindungen des Menschen, Jena, Gustav Fischer, 1931. Amprino, R., and Bairati, A.: Studi sulle trasformazioni delle cartilagini dell-uomo nell'accrescimento e nella senescenza, Ztschr. f. Zellforsch. u. mikr. Anat. **21**:448 (Aug.) 1934. Gatta, R.: Osservazioni sulla struttura e sullo sviluppo della sinfisi pubica, Arch. ital. di anat. e di embriol. **33**:813, 1934.

of these sections was placed flat and roentgenographed in the superior-inferior plane. After this, one or more portions removed from the middle third of the specimen were fixed in Helly's solution,[1a] decalcified in 5 per cent nitric acid, embedded in paroxylin (celloidin) and stained with hematein and eosin.

DEVELOPMENT OF THE SYMPHYSIS PUBIS

There has been a large amount of embryologic and anatomic work on this question.[2] The investigations have been carried out in man and in many species of animals. The results of such studies and the more or less accepted anatomic facts can be briefly summarized. Each pubic bone is derived from a center of ossification developing in cartilage. The cartilage plates meet in the midline of the body to form the symphysis pubis. Anteriorly, undifferentiated mesenchyme derived from the anterior ligament binds the two cartilage plates. This mesenchymal

Age Groups and Sex Groups Represented by the Specimens Studied

Age, Years	Males	Females		Specimens
		Nulliparas	Multiparas	
0- 5	4	3	..	7
6-10	2	3	..	5
11-20	6	..	..	6
21-30	..	2	1	3
31-40	7	3	2	12
41-50	7	2	3	12
51-60	8	7	4	19
61-70	5	2	2	9
71-80	2	..	..	2

tissue can be transformed into hyaline cartilage; in fact, this regularly occurs in some animals. In adult rats, however, there is no intervening tissue in the midline between the cartilaginous elements of the pubic

1a. Helly's solution consists of: potassium bichromate, 2.5 Gm.; mercury bichloride, 5 Gm., and water, 100 cc. Add to 90 cc. of this solution 10 cc. of a 40 per cent solution of neutral formaldehyde solution, U.S.P., just prior to the fixation of the tissues.

2. Barlow, M.: Peculiarities of the Pelvis in the Cow, Month. J. M. Sc. **18**:83, 1854. Humphry, G. M.: A Treatise on the Human Skeleton, Cambridge, Macmillan & Co., 1858. Flower, W. H.: Osteology of the Mammalia, London, Macmillan & Co., 1885. Bardeen, C.: Studies of the Development of the Human Skeleton, Am. J. Anat. **4**:265 (May) 1904-1905. Franz, K.: Zur Entwicklung des knöchernen Beckens nach der Geburt, Beitr. z. Geburtsh. u. Gynäk. **13**:12, 1908-1909. Adair, F. L.: The Ossification Centers of the Foetal Pelvis, Am. J. Obst. & Gynec. **78**:175 (Aug.) 1918. Dorland, W. A. N., and Hubeny, M. J.: The X-Ray in Embryology and Obstetrics, London, Henry Kimpton, 1926. Dawson, A. B.: Further Studies on the Epiphyses of the Albino Rat Skeleton, with Special Reference to the Vertebral Column, Ribs, Sternum and Girdles, Anat. Rec. **34**:351 (Feb.) 1927.

bones[3] (fig. 1 *A* and *B*). In man, the fibrous tissue between the cartilaginous formations is incompletely transformed into hyaline cartilage. This communicating mesenchyme is subject to conversion, in some degree, into fibrous tissue or into fibrous or hyaline cartilage. Very early, even in intra-uterine life, clefts may be seen in that interstitially placed connective tissue.[4] This was noted in the posterior portion of the symphysis pubis of a human fetus (at 4 months; fig. 1 *C*). In the newly born infant, the undifferentiated mesenchyme appears as an insertion of fibrous tissue or fibrocartilage between the cartilaginous portions of the pubic bones. This tissue may or may not contain the clefts mentioned. It may therefore be inferred that there is no basis for considering the pubic symphysis as a true joint region (fig. 1 *D*).

The results of the present studies will be presented as (*A*) appearances in the young and (*B*) appearances in the adult.

GROUP A (AGES 1 TO 17 YEARS)

Three typical specimens will be described. They illustrate clearly the characteristic roentgenographic, gross and histologic observations in the young.

Roentgenographic examination of a specimen removed from the body of a 3 year old child plainly demonstrated the beginning of a dentate appearance in the symphyseal ends (fig. 2 *A*). This was a consistent observation in all roentgenograms of pubic bones removed from the bodies of infants.

On gross examination, it was found that the pubic bones in the region of the symphysis were bound together by four external ligaments (superior, inferior, anterior and posterior). The application of considerable force in the anterior direction relaxed the anterior ligament, although there was marked resistance to motion in any of the other directions. On section of the various specimens in the anterior-posterior plane, the thick anterior ligament was clearly disclosed, with its projection between the end-plates of the pubic bones. The growth zones at the symphyseal ends of the pubic bones presented a slightly serrated surface (fig. 2 *B*). The gross appearance was thus entirely in conformity with that observed on roentgenographic examination. The fibrous tissue between the cartilaginous ends of the pubic bones was recognized by roentgenographic examination as a dense linear shadow (fig. 2 *C*).

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3. As Chapman points out, the symphysis pubis may be entirely absent in rodents. As a substitute, the right rectus muscle is attached to the left pubic bone and the left rectus to the right pubic bone (Chapman, R. N.: A Study of the Correlation of the Pelvic Structure and the Habits of Certain Burrowing Mammals, Am. J. Anat. **25**:185 [March] 1919).

4. Ruth, E. B.: The Study of the Development of the Mammalian Pelvis, Anat. Rec. **53-54**:207 (July) 1932.

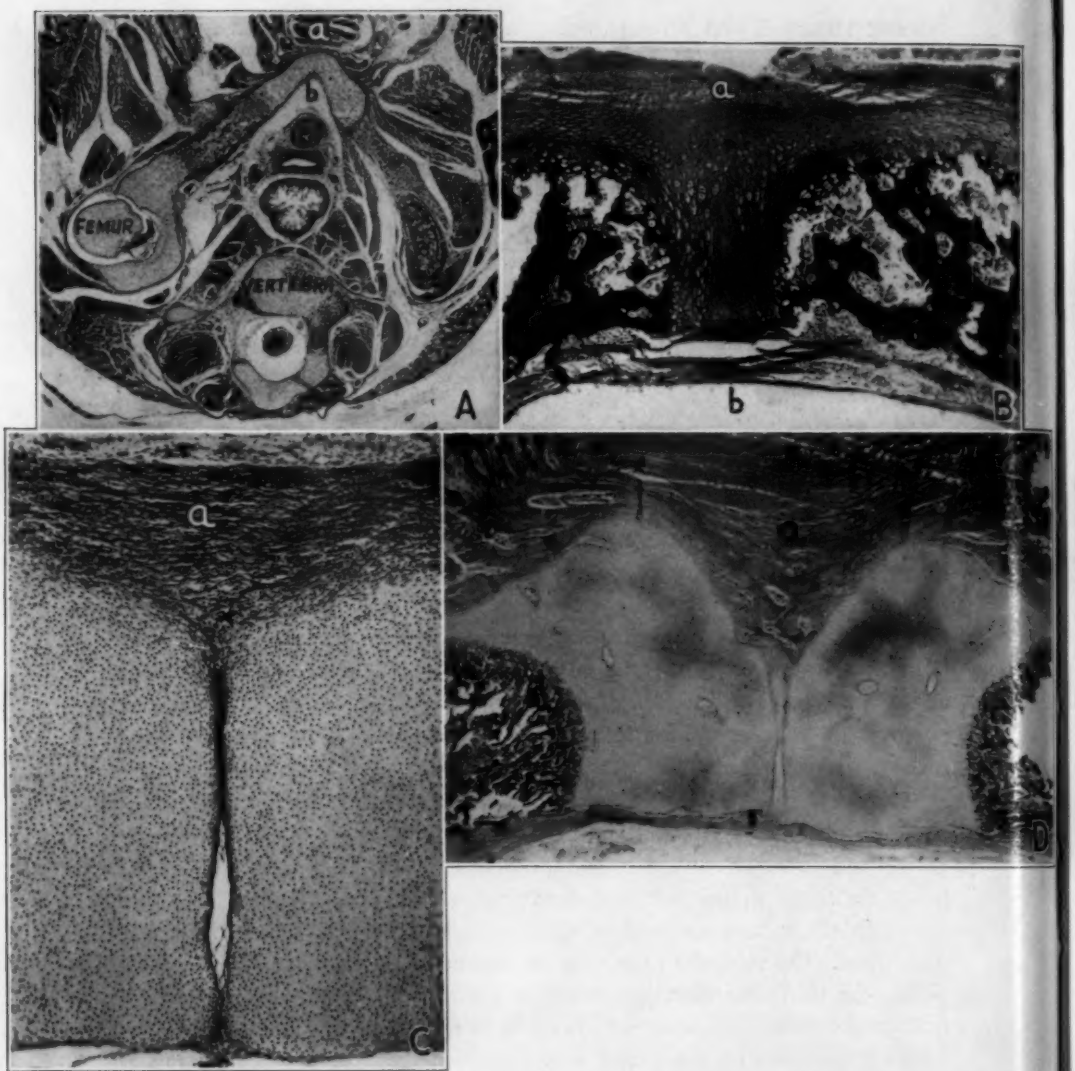

Fig. 1.—*A*, photomicrograph of the region of the symphysis pubis in a new-born rat; reduced from × 80. Note the uniform plaque of hyaline cartilage which binds both pubic bones; *a* is the anterior and *b* the posterior ligament. The latter faces the peritoneal cavity. *B*, photomicrograph of a similar region in an adult rat; reduced from × 80. Note the pubic bones and their respective growth plates. A wide layer of hyaline cartilage connects them; *a* is the anterior and *b* the posterior ligament. *C*, photomicrograph of the symphysis pubis from a human fetus at approximately 4 months; reduced from × 80. Note the anterior ligament (*a*) and its invagination between the two embryonic cartilage plates. A cleft is present in the posterior portion of this projection. *D*, photomicrograph of the symphysis pubis from a new-born infant; reduced from × 8. Note the anterior ligament (*a*) and its invagination between the two pubic cartilage end-plates.

On histologic examination, the hyaline cartilage plates of the specimen (from the 3 year old child) were quite thick and were bound together by a layer of fibrous tissue. The latter extended about seven eighths of the anterior-posterior distance of the symphysis pubis. The remaining (posterior) one eighth was occupied by a semilunar bridge of

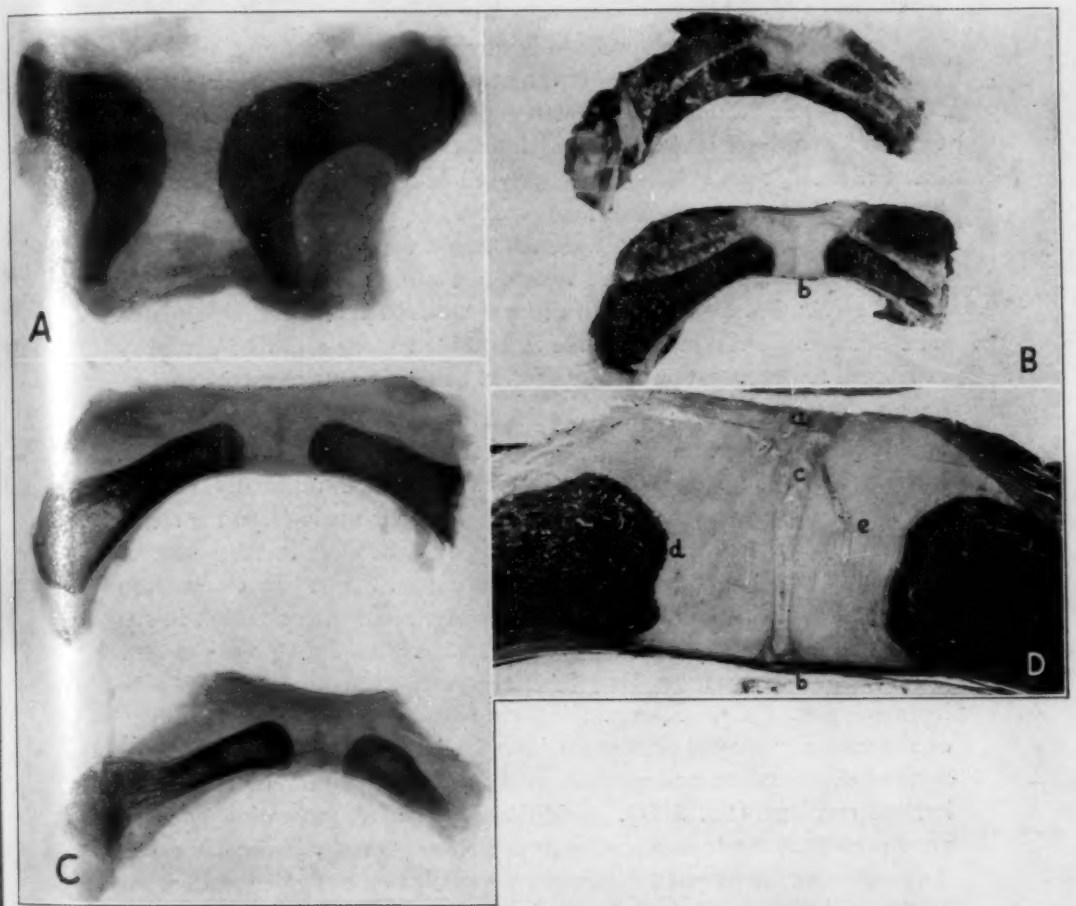

Fig. 2.—*A*, roentgenogram of the pubic bones from a 3 year old girl, showing the beginning of a serrated appearance of the growth plates. *B*, photograph of two slivers sectioned from the same specimen in the anteroposterior plane, showing the wide pubic cartilages separated by a fibrous layer. Note the thin posterior ligament (*b*). *C*, roentgenogram of these two slivers, showing the projection of the anterior ligament between the two hyaline cartilage plates. *D*, photomicrograph of one of the slivers; reduced from × 6. Note the anterior ligament (*a*) and its projection (*c*) between the hyaline cartilage plates. The growth zones are serrated in appearance (*d*). The posterior ligament (*b*) is thin. Note the blood vessel (*e*) in the hyaline cartilage.

fibrocartilage. The aforementioned fibrous invagination merged with the contiguous hyaline cartilage and contained numerous blood vessels, some of the vessels penetrating the cartilage. Small irregular-shaped spaces were noted, but no genuine joint cavity was discernible. On the endosteal surface, the posterior cortices were lined by numerous osteoblasts. The subperiosteal surface showed Howship's lacunae and osteoclasts (fig. 2 *D*).

On examination of a specimen removed from the body of an 8 year old boy, nothing remarkably different was noted. A similar dentate shadow was apparent at the growth zone in the roentgenographic plate. Gross examination revealed turgid interpubic fibrous and fibrocartilaginous tissues. Small clefts were observed in these tissues, most marked in ventral portions of sections taken from the upper part of the specimen. Microscopic examination again demonstrated the undulating appearance of the growth zone, with blood vessels in the interpubic tissues. A few vascular channels extended into the contiguous hyaline cartilage plates. The numerous clefts noted in the interpubic tissues could be definitely attributed to degeneration and separation of the fibrous elements.

The third specimen under consideration came from the body of a boy 14 years of age. Death had been caused by a miliary tuberculosis. Roentgenographic study of the specimen revealed unusually dentate outlines at the growth zones (fig. 3 *A*). Sections made from the specimen showed on gross examination a thin midline cleft in the posterior two thirds of the interpubic fibrous tissue (fig. 3 *B*). In comparison with the two specimens described previously, relative diminution in the width of the hyaline cartilage end-plates was evident. The thickness of the anterior ligament was from four to six times that of the posterior ligament (fig. 3 *C*). Microscopic examination confirmed the presence of the irregular-shaped cleft in the interpubic tissues, the consequence of marked degeneration and necrosis of both interpubic fibrous tissues and hyaline cartilage (fig. 3 *D*). Additional foci of degeneration likewise were present in the hyaline cartilage as well as several microscopic cysts. An interesting feature of this specimen was the unusually deep extension of the thick anterior ligament into the hyaline cartilage, creating a relative increase of fibrocartilage in the symphysis pubis. There was no great extent to the penetration of the thin posterior ligament into the hyaline cartilage plates. The considerably greater thickness and greater compactness of the posterior cortex of the pubic bones may perhaps be explained as compensation. The anterior cortex was partly composed of immature bone; its marrow spaces, for the most part, were in direct contact with the anterior ligament.

GROUP B (ADULTS)

In the usual development of pubic bones "retrogressive epiphyses" appear after puberty. Todd [5] found these bony nodules (not true centers of ossification) on the superior and inferior aspects of the ventral surface of each pubic bone near the symphyseal face (fig. 4). Although highly erratic in their mode of development, they generally tend to meet and fuse along the ventral border of each pubic bone. Changes, such as

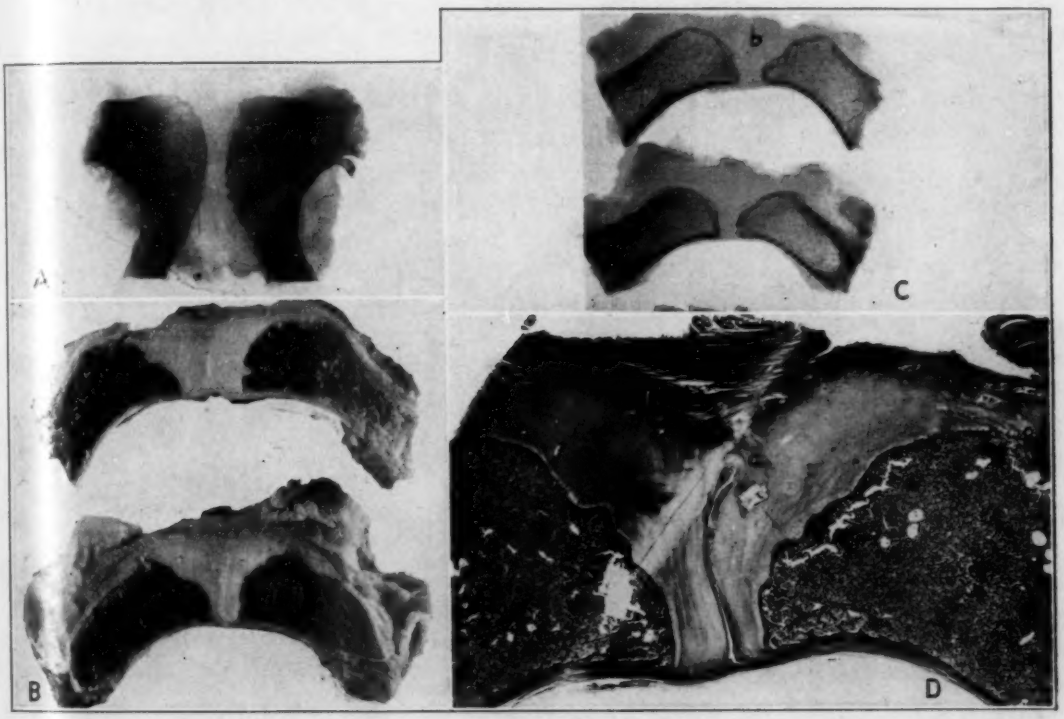

Fig. 3.—*A*, roentgenogram of the pubic bones removed from a 14 year old youth showing an unusual serrated outline at the growth zones. *B*, photograph of slivers sectioned in the anteroposterior planes, from the specimen shown in *A*, revealing the fibrous interpubic sheath. In it is a very thin cleft. The anterior ligament is marked *a*. *C*, roentgenogram of these slivers disclosing the dentated configuration of the growth plates. Note the greater width of the interpubic tissue in its anterior portion (*b*). *D*, photomicrograph of one of the slivers; reduced from × 5. It demonstrates the cleft in the interpubic tissue. Note the invasion of the hyaline cartilage by the anterior ligament (*a*).

the cessation of endochondral ossification, result in the formation of a subchondral bony plate. Its appearance, varying widely, is sometimes

5. Todd, T. W.: Age Changes in the Pubic Bone, Am. J. Phys. Anthropol. **3**:285 (July) 1920; **4**:1 (Jan.) 1921; **4**:333 (Oct.) 1921; **14**:255 (April) 1930.

notched, sometimes straight and often dentate.[6] Todd discerned ten different stages through which the pubic bone of the adult passes in its development.

When specimens of the symphysis pubis removed at autopsy from adults were selected for study, such as were apparently involved by disease (except arthritis) were excluded.

On gross examination it was possible to produce slight motion in several specimens removed from the bodies of adult males and nulliparas. However, only when the pubic bones were forced anteriorly was motion obtained. Widening the interpubic space was almost impossible.

There was no uniformity in the roentgenographic appearance of the subchondral zones; in some, traces of the original undulated structure were still distinguishable (fig. 5 *A*); in others, flat sclerotic zones only were seen. The latter observation was made particularly in specimens

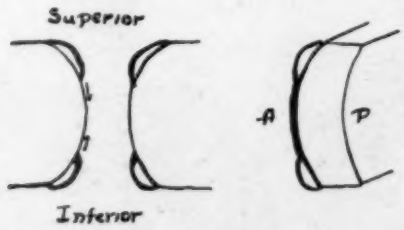

Fig. 4.—A schematic representation of the superior and inferior "retrogressive epiphyses" of the pubic bones. They attempt to meet and fuse along the anterior surfaces: *A*, anterior; *P*, posterior.

taken from adults of from 40 to 50 years (fig. 6). Some porosis of the subchondral zones was manifested in the greater portion of specimens taken from adults past 50 years. Definite sclerotic subchondral zones persisted in several specimens taken from adults between 50 and 80 years of age (fig. 7 *A*). In a number of roentgenograms of group B, the ventral rampart or anterior corner—the ledge of bone formed along the anterior surface of the pubic bone close to the symphyseal face (described)—was clearly demonstrated (fig. 5 *A*).

In macroscopic examination of serial sections, the clearly visible anterior ligament showed its decussating fibers deeply injected into the

6. Krauss, F.: Ueber Symphysensprengung, Zentralbl. f. Chir. **57**:134, 1930. Chamberlain, W. E.: The Symphysis Pubis in the Roentgen Examination of the Sacro-Iliac Joint, Am. J. Roentgenol. **24**:621 (Dec.) 1930. Sack, G. M.: Zur Pathologie der Symphyse, Röntgenpraxis **5**:566 (Aug.) 1933. Burman, M. S.; Weinkle, I. N., and Langsam, M. J.: Adolescent Osteochondritis of the Symphysis Pubis, J. Bone & Joint Surg. **16**:649 (July) 1934.

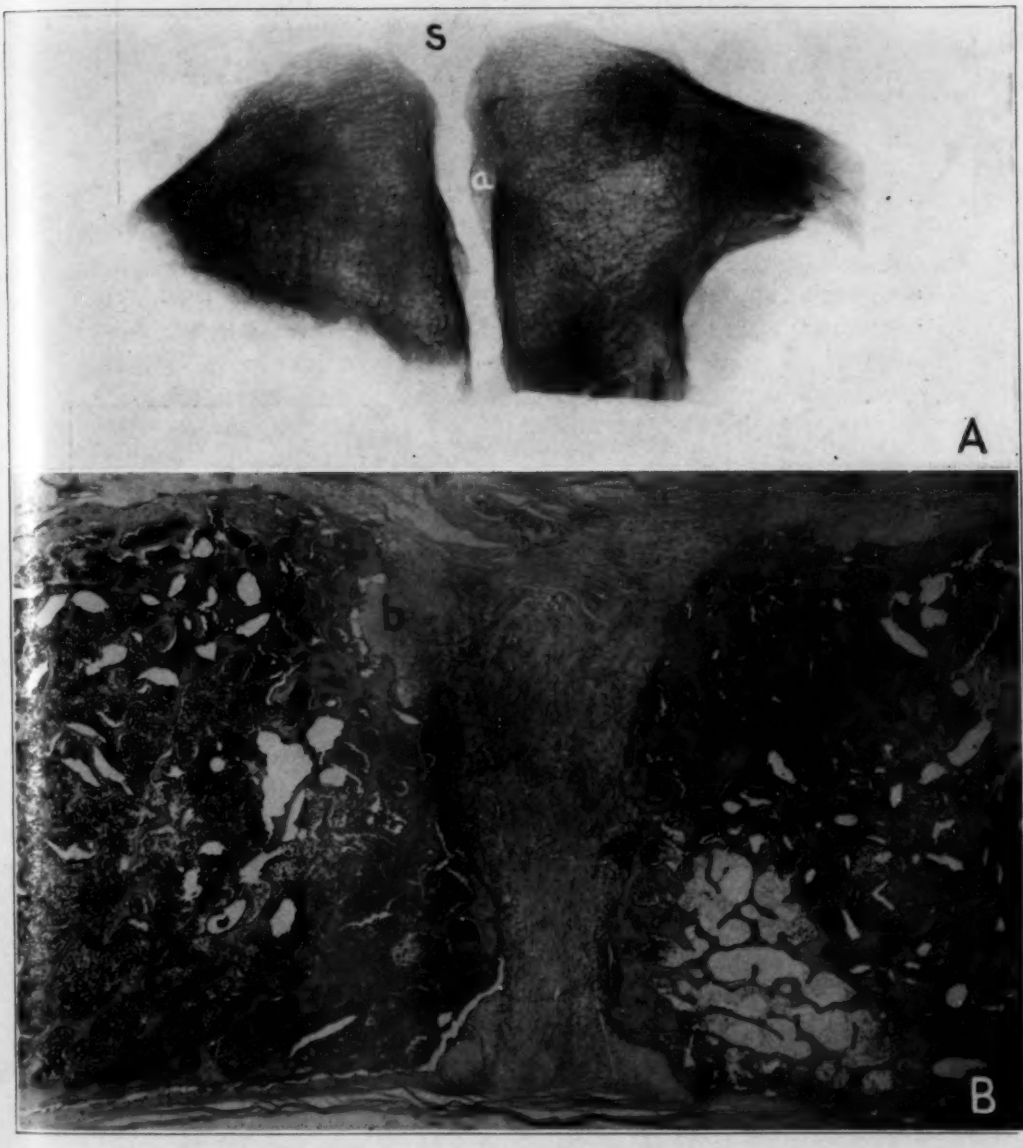

Fig. 5.—*A*, roentgenogram of the pubic bones from a man 48 years old, revealing that the ventral rampart or anterior corner (*a*) is serrated in appearance. The superior surface of the symphysis pubis is labeled *s*. *B*, photomicrograph of a section prepared from a sliver cut in the anteroposterior plane; reduced from × 5. It reveals no fissures in the symphysis pubis. Note the absence of the anterior corner in the left pubic bone (*b*).

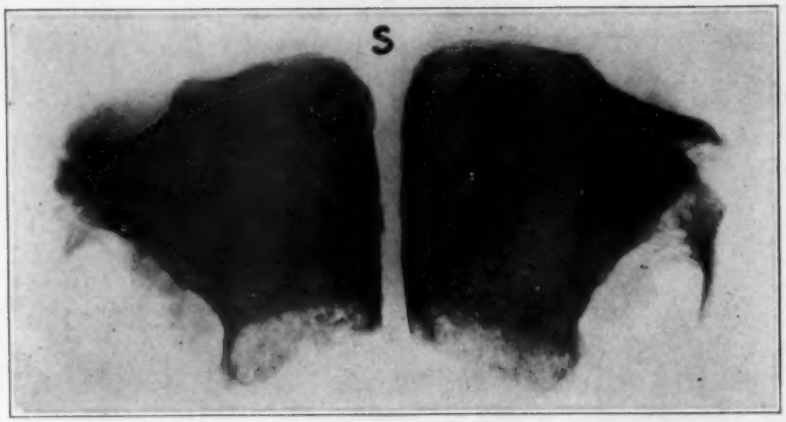

Fig. 6.—Roentgenogram of the pubic bones removed from a man 40 years of age, showing dense subchondral zones. Very little irregularity is noted. The superior surface of the symphysis pubis is marked *s*.

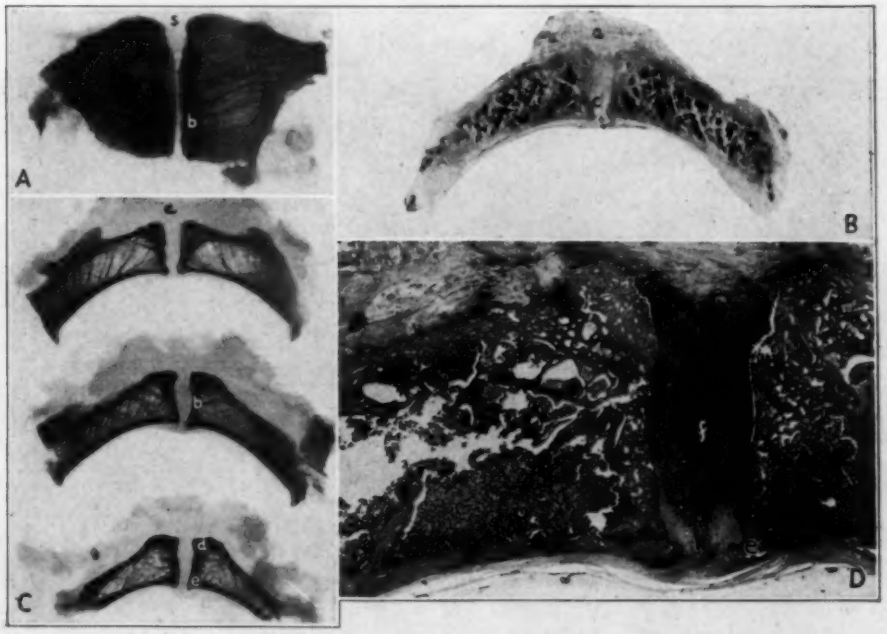

Fig. 7.—*A*, roentgenogram of the pubic bones removed from a man 53 years old, disclosing marked sclerosis of the subchondral regions (*b*). The superior surface is labeled *s*. *B*, photograph of a sliver cut from the specimen shown in *A* in an anteroposterior plane, exhibiting the thick anterior ligament (*a*). The fibrocartilaginous symphysis pubis contains a cleft in its posterior aspect (*c*). *C*, roentgenogram of several slivers cut from the same specimen in an anteroposterior plane, demonstrating the thick anterior ligament (*a*). The sclerotic subchondral zones (*b*), the anterior corners (*d*) and the prominent posterior corners (*e*) are clearly seen. *D*, photomicrograph of the section prepared from the middle sliver; reduced from × 5. It reveals very thick subchondral zones (*b*) and anterior corners (*d*). The posterior corners (*e*) are prominent. Note that the interpubic tissue consists mainly of fibrocartilage (*f*). *A* few fissures are present in it.

interpubic cartilage. The ligament was thicker than the posterior one (fig. 7 *B*), except in specimens taken from multiparas.

Contrary to the appearance in the young, in whom the end-plates of the pubic bones are composed of thick zones of hyaline cartilage, only small segments of such cartilage appeared in the adult group. Those seen were in the anterior or the posterior portions of the symphysis pubis close to the pubic bones. The rest of the cartilage end-plate which had been hyaline was transformed into fibrocartilage or was extremely fibrillated. The cartilaginous end-plates varied in color from brownish yellow to distinct yellow.

In group B, again, the irregular clefts were manifest in many of the specimens. They appeared either in the middle of the interpubic tissue or close to the subchondral zones, extending from the anterior to the posterior ligament and from the superior to the inferior ligament. Some were observed only in the anterior, the middle, or even the posterior third of the symphysis pubis. Sometimes, the latter clefts were extensions of those in the anterior or the posterior ligament. Some of these clefts were wide enough to admit the bulbous head of an ordinary pin; several clefts had irregular branches.

Roentgenographic examination of serial sections demonstrated clearly the subchondral zones as well as the anterior corners arising from the "retrogressive epiphyses" (fig. 7 *C*). In some cases, the subchondral zones of the right and left pubic bones were not parallel to each other. They diverged toward the anterior cortices. Roentgenographic examination of a few sections disclosed small cystlike shadows varying in size from 2 to 6 mm. in or near the subchondral zones (fig. 8 *A*). Histologic studies revealed them as either cartilaginous herniations or discrete islands of cartilage (fig. 8). Also present, were small osteophytes projecting from the subchondral zones into the symphysis pubis (*c*, fig. 9 *A*). Bony projections were observed in some to be near or at the posterior corners, i. e., the junction of the posterior cortex and the symphyseal face.

Histologic Studies.—Interpubic Tissue: Each section studied in group B had definite fibrillation of some parts of the hyaline cartilage, usually with marked fibrous invasion of the remaining hyaline cartilage (fig. 7 *D*). This demonstrated its cause to be the expansion of the anterior ligament with its fibrous projection at the expense of the hyaline cartilage. Close to the subchondral bone, small remnants of hyaline cartilage remained intact. The clefts were again observed in the interpubic tissues. In specimens from males (fig. 9 *C*) they were rather limited, and several such specimens were totally without any evidence of fissures (fig. 5 *B*). Irregular fissuring was observed to a slightly greater extent in specimens obtained from nulliparas (fig. 8 *B*).

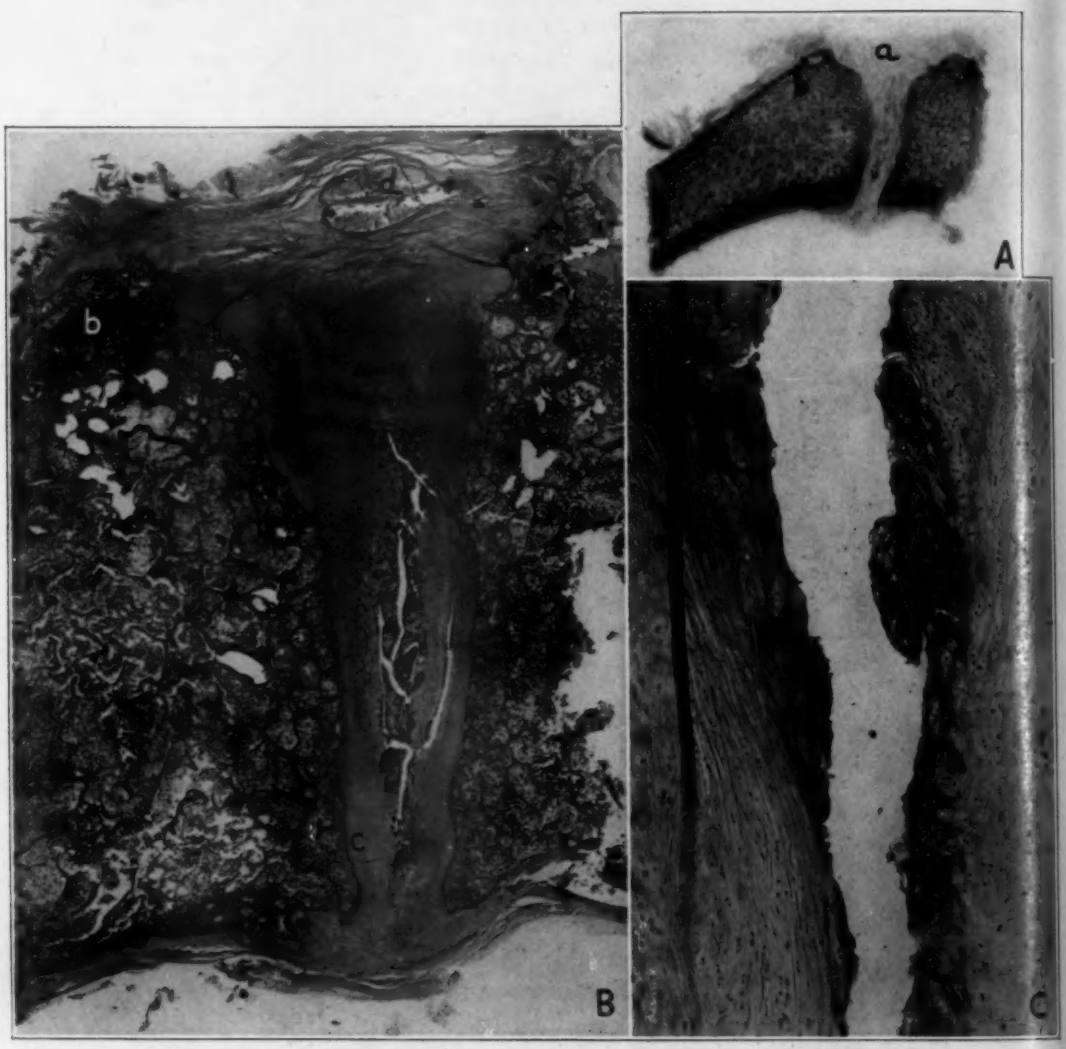

Fig. 8.—*A*, specimen removed from a nullipara 55 years old. The sliver was sectioned in the anteroposterior plane. The anterior portion is marked *a*. The roentgenogram demonstrates a cystic space in the anterior corner marked *b*. *B*, photomicrograph of the histologic section prepared from the sliver; reduced from × 6. The cyst (*b*) is seen to be the result of a cartilaginous herniation. There is irregular fissuring of the symphysis pubis. A small segment of it is still of the hyaline variety (*c*). A degenerative cyst may be noted in the anterior ligament (*d*). *C*, a high power photomicrograph reveals that the cleft in the fibrosed pubic cartilage is bordered by degenerated and necrotic fibrillar cartilage. Note the island of necrotic cartilage in the fissure; reduced from × 100.

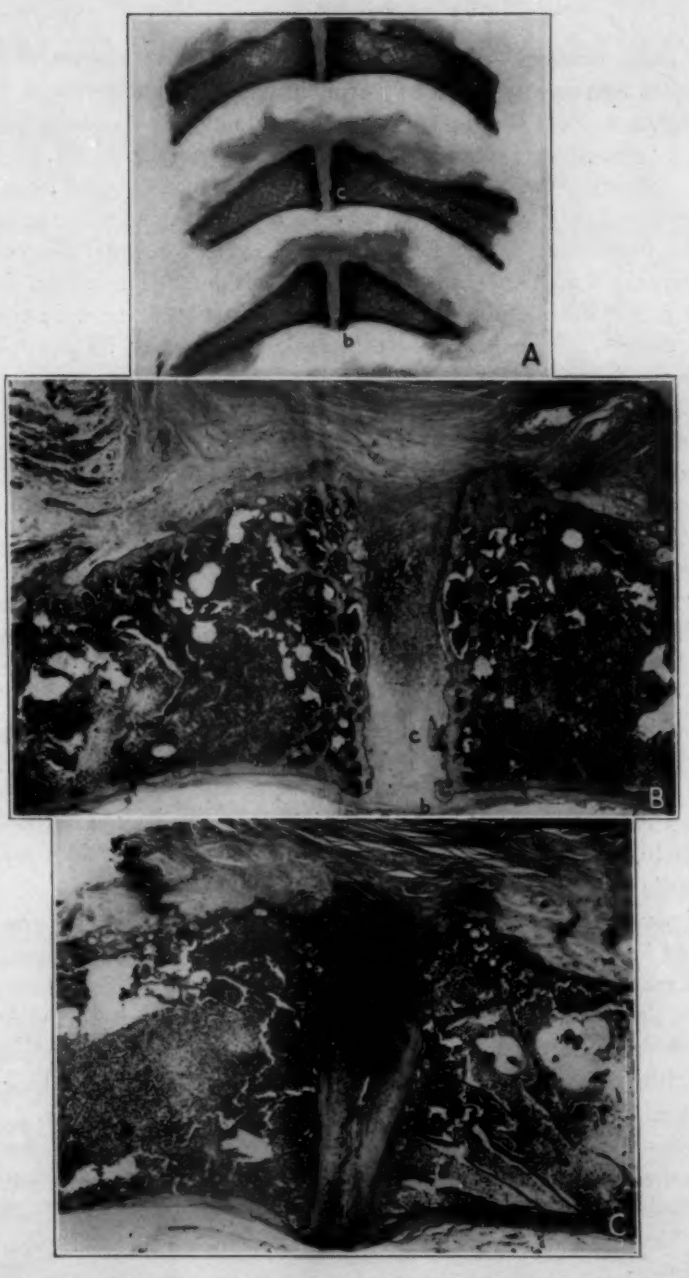

Fig. 9.—*A*, specimen from a man 41 years old. The slivers were cut in the anteroposterior plane. The roentgenogram reveals prominent posterior corners (*b*) as well as an osteophyte projecting into the interpubic tissue (*c*). *B*, photomicrograph of the middle sliver; reduced from × 5. Note the osteophyte in the interpubic tissue (*c*), also that at the posterior corner (*b*). *C*, photomicrograph of a section prepared from a sliver cut in the anteroposterior plane; reduced from × 5. The specimen is from a man 55 years old. A few fissures may be seen in the fibrocartilaginous symphysis pubis.

No true synovial membrane or lining was apparent at the borders of the clefts and fissures in the interpubic tissues. These cracks revealed themselves as degenerative effects, the tissue at their borders evidencing focal or generalized degeneration and necrosis (fig. 8 *C*). Cartilage some distance from the fissures gave like evidence of mild or advanced degeneration (demonstrated by poor staining of nuclei and edema of the matrix, mucoid or fatty in nature). Groups of regenerating cartilage cells appeared in the border tissue, while islands of necrotic, living or regenerating cartilage and amorphous detritus were seen in the interpubic tissues. In specimens from male adults over 50 years of age, where no fissures were discernible, the interpubic area consisted solely of fibrocartilage, occasionally with increased amounts of mucoid that gave a semblance of hyaline cartilage.

Subchondral Zone: Compact lamellar bone constituted the structure of the subchondral bony plates in the greater number of sections. In several, however, from adults over 50 years of age, porosis and focal necrosis existed, while in a few there were inclusions or herniations of either fibrous or hyaline cartilage. When the latter was present there was a slight appearance of endochondral ossification. The interpubic tissues were invaded by vascular channels of subchondral origin. Therefrom ensued the formation of bony osteophytes and marrow spaces in the interpubic tissues (fig. 9 *B*). Osteophytes may similarly be situated in the provisional zone of calcification at either the anterior or the posterior corners of the pubic bones.

Cortices: Posteriorly, the cortices were somewhat thicker than anteriorly. In some specimens taken from elderly adults, scattered areas of aseptic necrosis were occasionally noted.

Ligaments: The decussating fibers of the ligaments were deeply inserted in the interpubic tissues. Shorter fibers attached themselves to the corners, thus firmly fixing the pubic bones. Like the interpubic tissues, the ligaments may suffer degenerative changes with the resultant occurrence of clefts and cysts (fig. 8 *B*). The cysts may contain necrotic ligamentous tissue and occasionally islands of either dead or living cartilage.

Specimens from Multiparas: It is recognized that pregnancy induces alterations in the appearance of the pubic bones and of the symphysis pubis. Osteoclastic resorption of the symphyseal ends of the pubic bones has been observed in pregnant guinea-pigs.[7] Investigators

7. Todd, W. T.: The Pubic Symphysis of the Guinea Pig in Relation to Pregnancy and Parturition, Am. J. Anat. **31**:345 (March) 1923. Kawata, S.: On the Morphological Changes of the Symphysis Pubo-Ischiadica in the Guinea Pig During Pregnancy, Folia anat. japon. **2**:370, 1924. Eymer, H., and Lang, F. J.: Anatomische Untersuchungen der Symphyse der Frau im Hinblick auf die

heretofore have demonstrated that the adult male guinea-pig has a cartilaginous, and the adult female a fibrous, symphysis pubis. Repeated injections of theelin (or a similar product) into the castrated adult guinea-pig causes transformation of the symphysis from cartilaginous to fibrous. Additional injection of blood from a pregnant animal into the feminized guinea-pig induces relaxation of the fibrous symphysis pubis.[8] It has been successfully demonstrated that in the human being relaxation of the symphysis pubis occurs during pregnancy.[9]

Specimens taken from multiparas were easily differentiated as such. Their most obvious distinction lay in the thickness of the posterior ligament, which appeared as a massive rounded elevation on the posterior surface of the symphysis pubis (fig. 10*A*). Examination of the sections

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Geburt. und klinische Deutung der Befunde, Arch. f. Gynäk. **137**:866, 1929. Lang, F. J., and Haslhofer, L.: Changes in the Symphysis Pubis and Sacro-Iliac Articulations as a Result of Pregnancy and Childbirth, Arch. Surg. **25**:870 (Nov.) 1932. Bertin, E. J.: Separation of the Symphysis Pubis, Am. J. Roentgenol. **30**:797 (Dec.) 1933.

8. Hisaw, F. L.: The Corpus Luteum Hormone: Experimental Relaxation of the Pelvic Ligaments of the Guinea Pig, Physiol. Zoöl. **2**:59 (Jan.) 1929. Fevold, H. L.; Hisaw, F. L., and Meyer, R. K.: Isolation of the Relaxative Hormone of the Corpus Luteum, Proc. Soc. Exper. Biol. & Med. **27**:604 (March) 1930. Hisaw, F. L.: Physiology of the Corpus Luteum, in Allen, E.: Sex and Internal Secretions, Baltimore, Williams & Wilkins Company, 1932, p. 499. Pommerenke, W. T.: The Cyclic Relaxation of the Pelvic Ligaments in the Guinea Pig, Anat. Rec. **57**:361 (Nov.) 1933. Tapfer, S., and Haslhofer, L.: Hormonale Weiterstellung des Beckens im Tierversuch, Arch. f. Gynäk. **159**:313, 1935.

9. Duncan, J. M.: Researches in Obstetrics, Edinburgh, A. & C. Black, Ltd., 1868. Snelling, F. G.: Relaxation of the Pelvic Symphyses During Pregnancy and Parturition, Am. J. Obst. & Gynec. **2**:561 (Feb.) 1870. Eastman, T. B.: Rupture of the Symphysis Pubis, New York M. J. **83**:130 (Jan.) 1906. Tuley, H. E.: Rupture of the Symphysis Pubis in Labor, Am. J. Obst. & Gynec. **68**:852 (Nov.) 1913. Lynch, F. W.: The Pelvic Articulations During Pregnancy, Labor and the Puerperium, Surg., Gynec. & Obst. **30**:575 (June) 1920. Wishner, J. G., and Mayer, L.: Separation of the Symphysis Pubis, ibid. **49**: 380 (Sept.) 1929. Müller, W.: Röntgenologische Untersuchungen über die Symphyse Schwangerer, Zentralbl. f. Gynäk. (no. 112) **55**:999, 1931. Heyman, J., and Lundqvist, A.: The Symphysis Pubis in Pregnancy and Parturition, Acta obst. et gynec. Scandinav. **12**:191, 1932. Reis, R. A.; Baer, J. L.; Arens, R. A., and Stewart, E.: Traumatic Separation of the Symphysis Pubis During Spontaneous Labor, Surg., Gynec. & Obst. **55**:336 (Sept.) 1932. Boland, B. F.: Separation of the Symphysis Pubis, New England J. Med. **208**:431 (Feb.) 1933. Brouha, L.: Recherches sur la mobilisation de la symphyse pubienne chez le cobaye impubère, Compt. rend. Soc. de biol. **113**:406 (April) 1933. Barnes, J. M.: The Symphysis Pubis in the Female, Am. J. Roentgenol. **32**:333 (Sept.) 1934. Abramson, D.; Roberts, S. A., and Wilson, P. D.: Relaxation of the Pelvic Joints in Pregnancy, Surg., Gynec. & Obst. **58**:595 (March) 1934.

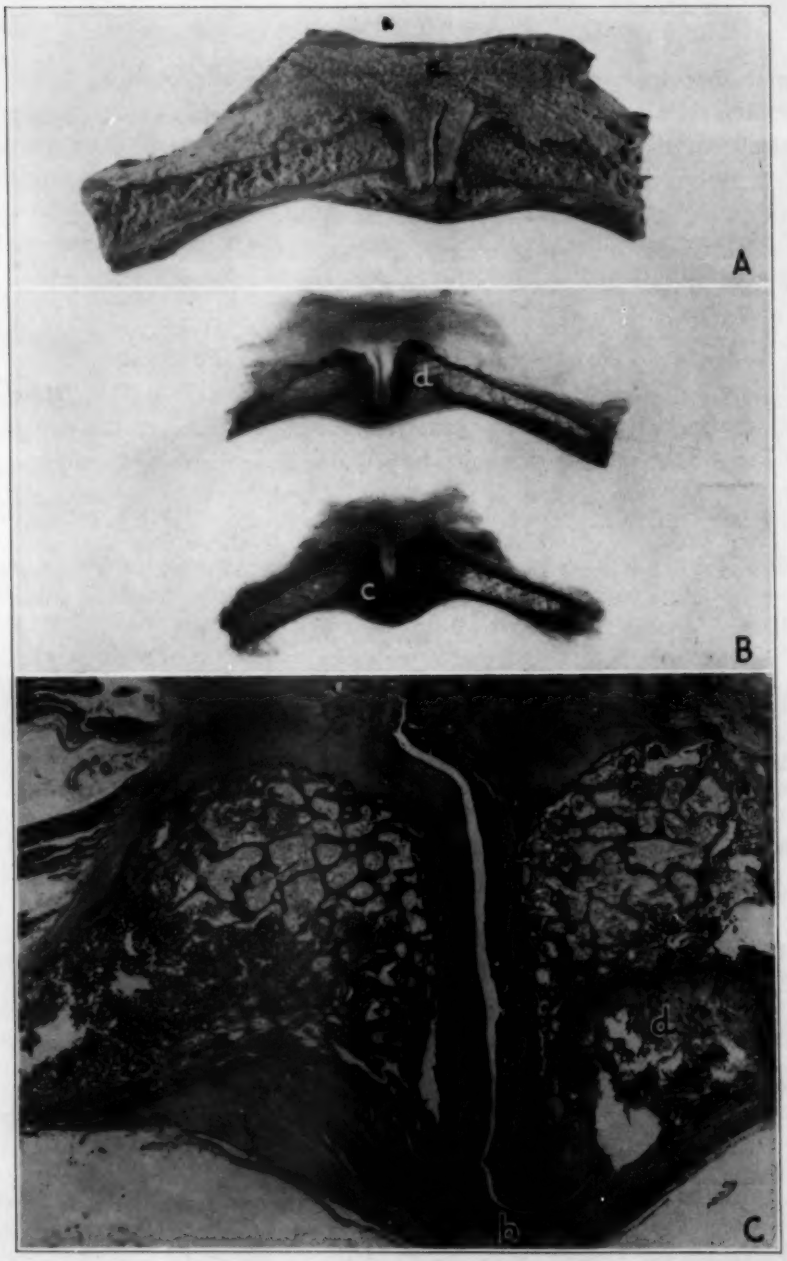

Fig. 10.—Photograph of a sliver from a specimen from a multipara 67 years old. It was sectioned in the anteroposterior plane. Note the thick posterior ligament (*b*) and the anterior ligament (*a*), which is continuous with the interpubic tissues and has in it a large fissure. *B*, roentgenogram of two slivers showing marked prominence of the posterior corners (*c*). Note the depressions in the posterior cortices for the insertions of the posterior ligament (*d*). *C*, photomicrograph of one of the slivers, revealing a large cleft in the symphysis pubis; reduced from × 6. This is continuous with those in the posterior ligament (*b*). The niche in the posterior cortex (*d*) is clearly seen.

again disclosed large irregular clefts in all portions of the interpubic tissues. The cartilage plates of the pubic bones were occasionally not attached to the pubic bones and posterior ligaments. In these cases their single point of fixation was the anterior ligament. Force exerted anteriorly produced considerable motion in some of the specimens. Roentgenograms of the sections showed exaggerated posterior corners. Some specimens demonstrated symmetrical depressions on the posterior cortices near the symphyseal surfaces. Within these areas were inserted the thickened posterior ligament (fig. 10 *B*). (One roentgenogram taken anteroposteriorly presented the posterior depression with a shadow like a cyst.)

Histologic examinations confirmed the gross findings. Marked degenerative changes and cleft formations had resulted in numerous cysts and channels throughout the ligaments and interpubic tissues. Many of the channels, particularly in the posterior ligament, were prolonged from one pubic bone to the other. The prominence of the thick posterior massive ligament proved to be due to the protuberance of the posterior corners in addition to an actual increase in the width of the posterior ligament with the protrusion of the posterior interpubic tissues (fig. 10 *C*). (This posterior mass is reserve tissue; relaxation during pregnancy probably permits further separation of the pubic bones.[10])

Pathologic Specimens: Evidences of pathologic processes were discernible in pubic bones when no trace or far less evidence of such processes could be found elsewhere in the skeleton. In this series of examinations, Paget's disease (figs. 11 *A* and *B*), idiopathic osteosclerosis, osteoporosis and metastatic foci of tumors (figs. 12 *A*, *B* and *C*) were some of the conditions encountered.[11] Roentgenograms showed that the subchondral bone at the symphyseal face may be destroyed by pathologic tissue. Thus, in roentgenograms, progressive obliteration of the shadow cast by the subchondral zone may give the clue to the presence of pathologic tissue.

SUMMARY AND CONCLUSIONS

Clefts in the interpubic tissues are due to degeneration; static forces may be responsible for their occurrence. Pregnancy causes relaxation and stretching of the ligaments and interpubic tissues, accentuating the

10. The relaxation of the thickened posterior ligament may permit contact with the pelvic organs. Also, the numerous channels in the posterior ligament may provide avenues for the spread of infection, possibly constituting the basis for the occurrence of puerperal osteomyelitis in the pubic bones.

11. Histologic examination of specimens involved in Paget's disease and in nutritional osteoporosis revealed vascularization and new cartilage formation in the interpubic tissue (fig. 12 *D*).

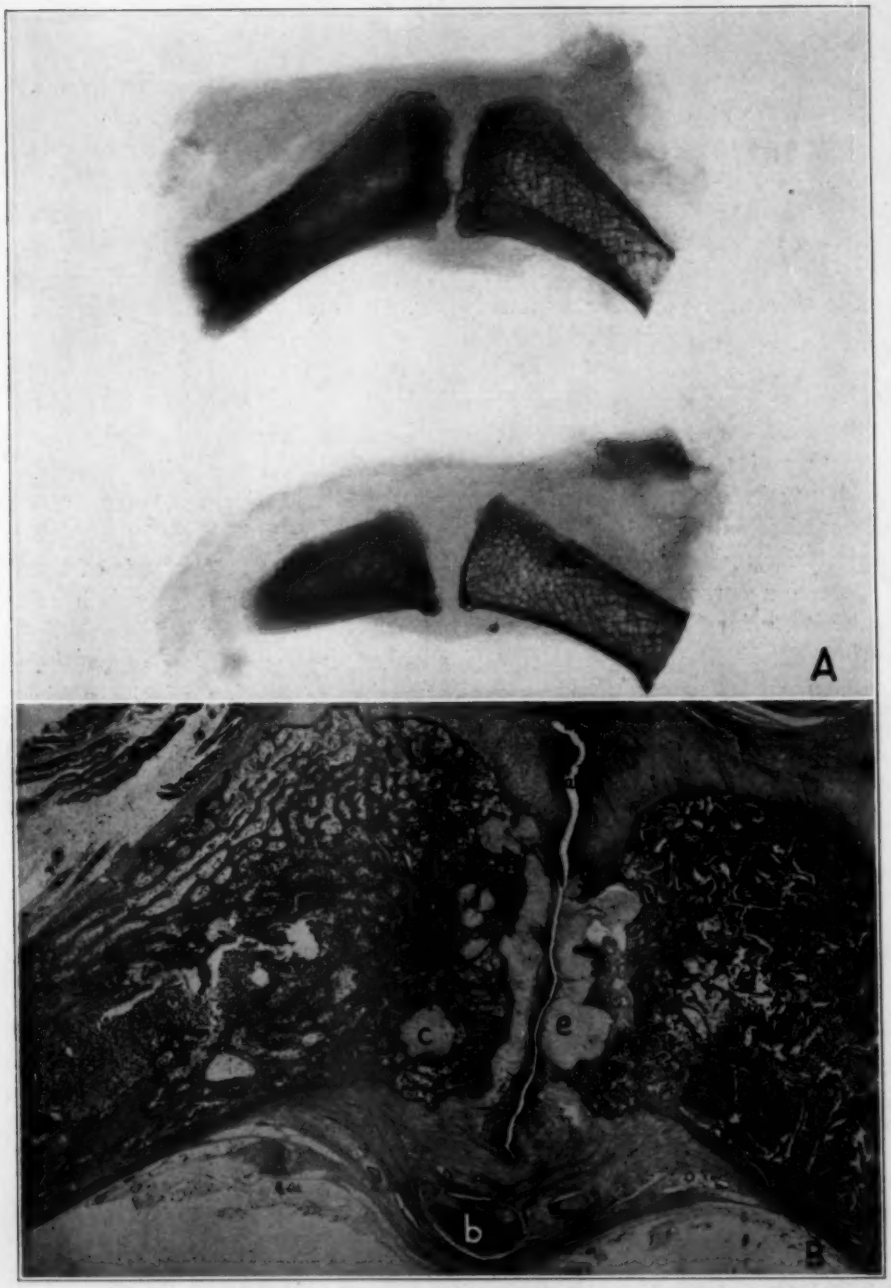

Fig. 11.—*A*, roentgenogram of slivers of a specimen from a multipara 53 years old. The slivers were sectioned from the specimen in an anteroposterior plane. Note the sclerosis of the right pubic bone—Paget's disease. *B*, photomicrograph of one of the slivers; reduced from × 5. It demonstrates a cleft in the interpubic tissue (*a*); a degenerative cyst in the thickened posterior ligament (*b*); numerous cartilaginous islands in the marrow (*c*); thickened cortices and closely set bone trabeculae (*d*) and newly formed pubic cartilage (*e*).

degenerative processes. In all specimens taken from adults, progressive (normal) degenerative manifestations could be observed.

Roentgenograms of the pubic bones taken from the young demonstrated the serrated appearance of the symphyseal face as normal. After puberty, growth may cause unusual conformation of the pubic face. Certain rare roentgenographic appearances may be noted owing to the presence of either cartilaginous herniations or inclusions.

The frequent demonstration of pathologic processes in the series studied emphasizes the importance of clinical and roentgenographic examination of the pubic region, especially on the slightest suspicion of

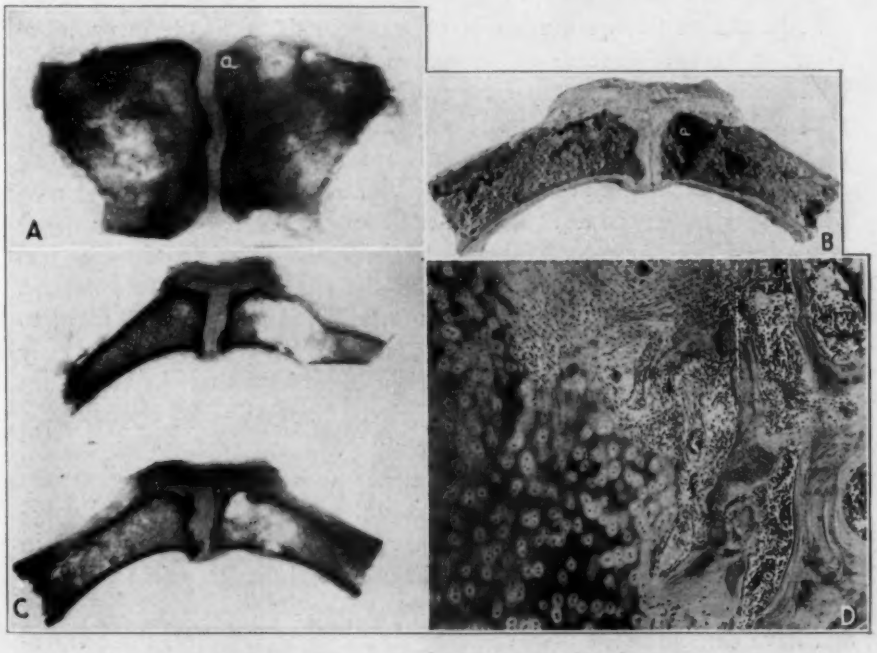

Fig. 12.—*A*, roentgenogram of a specimen from a nullipara 35 years old, who died from carcinoma of the stomach. Observe the punched-out areas in both pubic bones. A loss of the subchondral sclerosis is also apparent (*a*). *B*, photograph of a sliver sectioned from the specimen in an anteroposterior plane, which demonstrates invasion of the marrow, cortices and subchondral bone by a tumor (*a*). *C*, roentgenogram of two slivers revealing destruction of the cortices and trabeculae. Note the loss of the subchondral sclerosis (*a*). *D*, photomicrograph of a specimen from a man 39 years old; reduced from × 85. There was present marked nutritional osteoporosis. The photomicrograph shows a fibrous and vascular invasion of the pubic hyaline cartilage commencing from the subchondral region.

an osseous lesion. When postmortem examinations are limited in extent, extraction of the pubic bones for study is strongly advised; local and general osseous pathologic changes may thereby be elucidated.

SURVIVAL OF AN AUTOPLASTIC PARATHYROID TRANSPLANT IN A PATIENT WITHOUT PARATHYROID DEFICIENCY

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AND
ELLIOT C. CUTLER, M.D.
BOSTON

Autoplastic transplantation of parathyroid tissue has been practiced by a number of surgeons in connection with operations on the thyroid gland when one or more parathyroid glandules have been accidentally removed. This procedure has been advocated, particularly by Lahey, as a means of preventing the occasional occurrence of postoperative tetany. As far as we are aware, however, there has been no final proof of the survival of the transplant in these cases. Cattell [1] observed postoperative tetany in one of his patients in whom parathyroid tissue had been reimplanted between the fibers of the sternocleidomastoid muscle. The facts that the calcium content of the blood gradually returned to normal in the course of several weeks and that the tetany spontaneously and permanently disappeared were taken to indicate that the transplant had survived and was functionally active. There was no opportunity, however, to check this by microscopic study.

On the basis of Halsted's experimental work, it has been generally felt that these grafts will not survive unless there is a state of parathyroid deficiency following the transplantation, such as occurred in the patient observed by Cattell. We have studied the fate of parathyroid autografts in the dog under varying degrees of parathyroid deficiency and have demonstrated that, contrary to the general belief, these transplants will survive regardless of the physiologic need of the organism for parathyroid tissue.[2]

The clinical corroboration of this experimental finding has recently been observed.

A woman, aged 61, was subjected by us to total thyroidectomy on Feb. 4, 1933, because of intractable cardiac disease with severe angina pectoris and a previous history of coronary thrombosis. After removal the gland was carefully examined

From the Surgical Service of the Peter Bent Brigham Hospital.

1. Cattell, R. B.: Parathyroid Transplantation: Report of Autografts of Parathyroid Glands Removed During Thyroidectomy, Am. J. Surg. **7**:4 (July) 1929.

2. Shambaugh, P.: Autotransplantation of Parathyroid Gland in the Dog: An Evaluation of Halsted's Law of Deficiency, Arch. Surg. **32**:709 (April) 1936.

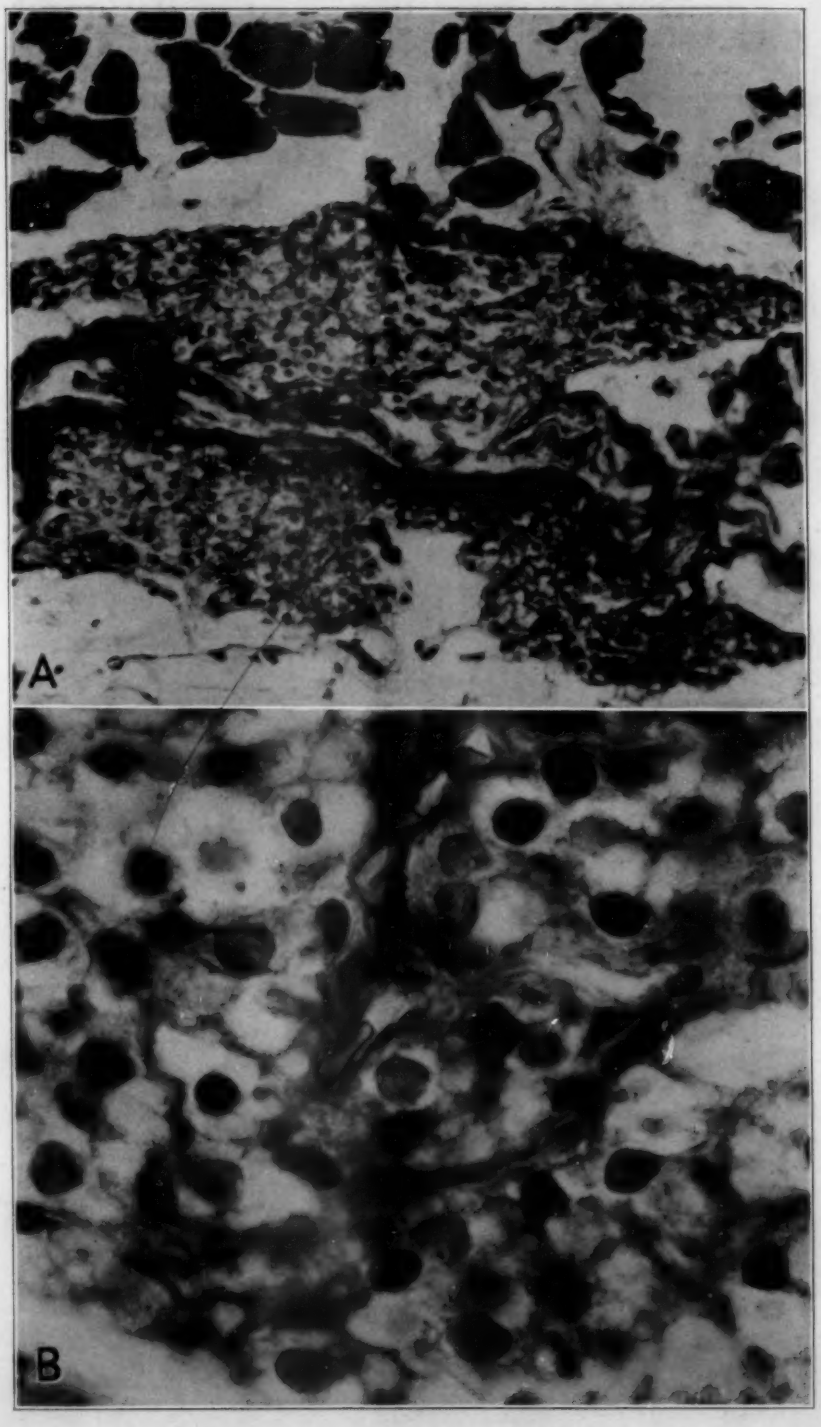

Fig. 1.—Photomicrograph of a parathyroid transplant: *A*, magnification × 300; *B*, magnification × 900.

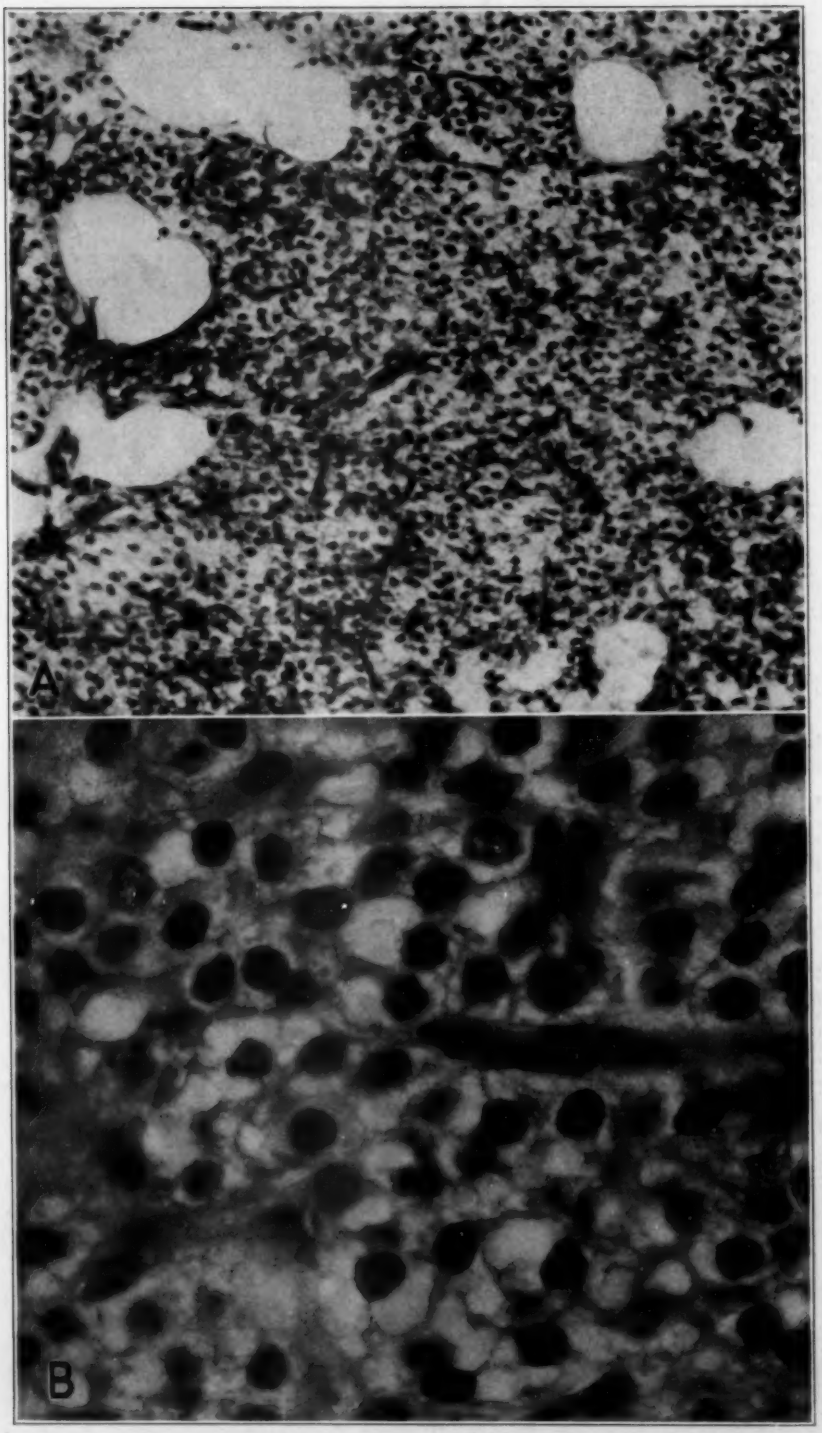

Fig. 2.—Photomicrograph of parathyroid glandule from which the graft was excised: *A*, magnification × 300; *B*, magnification × 900.

for parathyroid tissue, and one rather large glandule, measuring 5 by 8 mm., was found on the posterior surface of the right lobe near the lower pole. A small slice of this glandule was placed between the fibers of the left sternocleidomastoid muscle; the location was marked with a silk suture, and the remainder of the glandule was saved for microscopic identification. The patient made an uneventful recovery. Further examination of the thyroid gland showed a tiny piece of parathyroid tissue, 1.5 by 0.5 mm., on the posterior aspect of the right lobe near the upper pole. The left lobe showed no trace of parathyroid tissue.

The cardiac status of the patient was improved by the operation. She was completely relieved of anginal pain for five months, when a second attack of coronary thrombosis occurred. Following this there were infrequent and relatively mild anginal seizures, until death occurred with the third attack of coronary thrombosis on Sept. 18, 1935, two years and seven months after thyroidectomy.

Careful postmortem examination of the cervical region showed no thyroid tissue remaining, and there was no parathyroid tissue on the right side. On the left side the two parathyroid glandules were identified lying in their normal position. The muscle containing the transplant was excised, and on section the graft was recovered. There was virtually no fibrosis, and the cellular structure, when compared to the original glandule from which the graft had been taken, showed no alteration (figs. 1 and 2).

It can be assumed that the anatomic deficiency in parathyroid tissue at the time the graft was placed was 50 per cent. As far as can be determined, however, there was no physiologic deficiency. The patient showed no evidence of tetany at any time, and the calcium content of the blood did not fall postoperatively. In fact, there was a slight rise from the preoperative level of 11.2 mg. per hundred cubic centimeters to 11.5 mg. ten days after operation and 11.8 mg. fourteen days after operation. A final determination seven weeks after operation showed the calcium content to be 10 mg.

A coincidental finding was that the patient had Paget's disease of the bone involving the right ilium and, to a slight extent, the skull. We believe that this probably does not signify a parathyroid deficiency and therefore does not alter the conclusion that the parathyroid autograft survived in the absence of an urgent specific physiologic demand.

ARTHROSCOPIC EXAMINATION OF THE KNEE JOINT

REPORT OF CASES OBSERVED IN THE COURSE OF ARTHROSCOPIC EXAMINATION, INCLUDING INSTANCES OF SARCOID AND MULTIPLE POLYPOID FIBROMATOSIS

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We have been fortunate enough in the arthroscopic examination of eighty knee joints to observe a fair number of cases remarkable enough to merit individual report. One case of xanthoma of the knee was reported separately by Kling and Sashin.[1]

Arthroscopy has proved of great value in this group not only in ruling out other suspected disease processes but also in providing direct visual evidence of the existing disease, unusual as it may be. Visualization was usually good, and the pathologic picture was well demonstrated. While a proper diagnosis may not be made on the basis of visual evidence, enough is seen to indicate the proper therapeutic procedure. The ultimate diagnosis has sometimes rested on the microscopic observations. This group of cases supports an oft repeated statement that diseases of the knee joint can be as difficult and complex of diagnosis as any disease process within the chest or abdomen, despite the limited means of reaction of a joint.

SARCOID OF THE TENDON SHEATH AND JOINT

CASE 1.[2]—A girl, aged 8 years, has been under observation for more than two years. She was first admitted to the Hospital for Joint Diseases on Sept. 19, 1932, because of swelling of the knees, ankles, elbows and wrists. The disease process was of three years' duration, first beginning in the left knee and later involving the left ankle, left foot, right knee and right ankle, in this order. The elbows and wrists were involved last, about two years before admission. The patient felt weak, and her general health was only fair; yet she was able to be up and about throughout the development of the disease. In the few weeks before

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From the Hospital for Joint Diseases, the service of Dr. Leo Mayer.

1. Kling, D. H., and Sashin, D.: Hemorrhagic Villous Synovitis of the Knee Joint Due to Xanthoma, Arch. Surg. **30**:52 (Jan.) 1935.

2. An abstract of this case was included in an article by Burman, M. S.; Finkelstein, H., and Mayer, L.: Arthroscopy of the Knee Joint, J. Bone & Joint Surg. **16**:255, 1934.

admission she had noted a definite increase in the swelling of the knees, which had become painful. There had been occasional attacks of vomiting in the morning recently. There was no history of an important disease. The patient had had measles.

The child was definitely undersized and anemic and looked older than her age. She showed pronounced dental caries. Her heart and lungs were clinically normal. There was no splenomegaly or hepatomegaly. A few small shotty inguinal glands were felt, but otherwise no adenopathy was noted. The tonsils and adenoids were hypertrophied. Orthopedic examination revealed the hip joints

Fig. 1 (case 1).—A photograph of the patient at the time of admission in September 1932. Note the swelling of both knee joints and the thickening of the tendon sheaths about the ankles.

and shoulder joints to be free from the disease process which involved the other large joints. Both knees were swollen, owing both to the presence of effusion within the knee and to the definite thickening of the synovial lining. The measurements of the knees were as follows: right knee, 10¾, 11 and 8⅞ inches (27, 28 and 22 cm.), and left knee, 12, 12 and 9½ inches (30, 30 and 24 cm.), the measurements being taken at corresponding points in the suprapatellar region, over the patella and below the patella, respectively. Extension was full, but flexion was limited to 90 degrees. No heat or local spasm was present. Swelling

of the soft tissue was present on the inner aspect of both elbows. A roentgenogram made immediately after an injection of air into one of these masses showed it to be an extension of the synovial lining of the elbow and not a para-articular mass. Motion in the elbows was complete, and no heat was present about the joint. The masses on both wrists represented thickening of the tendon sheaths. There was a boggy and diffuse involvement of the common extensor tendons of the fingers, which formed a small mass on the dorsum of the hand. The

Fig. 2 (case 1).—A roentgenogram showing the cavitation in the olecranon process of the left elbow.

palmar surface of the hands was not involved. There was a slight but not significant restriction in the motions of the wrist. The fingers were considered to be normal. The ankles showed boggy swelling of the peroneal tendons laterally and involvement of the extensor sheath and the sheath of the tibialis posterior muscle. The sheath of the tibialis anterior muscle was not affected on either side. Slight limitation of ankle motion was present. The masses were not hot or red.

The diagnosis was uncertain. A pediatrician who was consulted diagnosed the condition as periarticular, symmetrical disease of infectious origin, possibly associated with the dental caries. Still's disease, which was considered, was ruled out by the absence of splenomegaly, the absence of adenopathy, the absence of irregular bouts of fever in the history (and indeed during the patient's stay in the hospital there was no significant variation in the temperature) and the significant freedom of motion in the swollen joints. The association of boggy masses

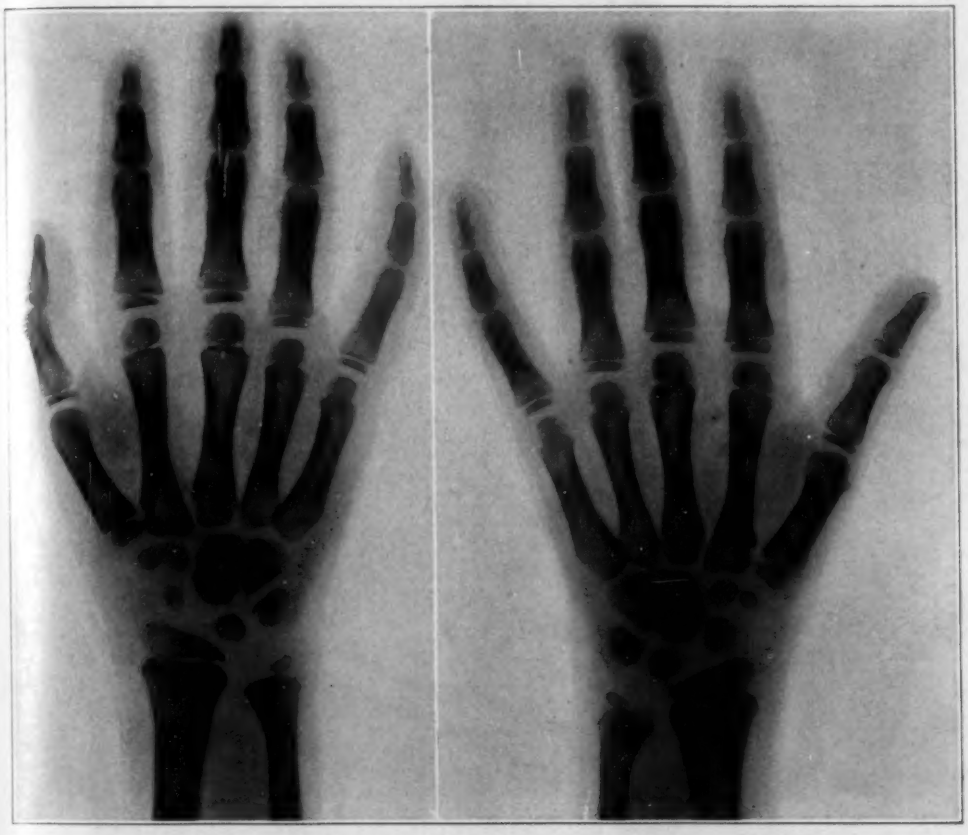

Fig. 3 (case 1).—A roentgenogram showing the trabeculated arrangement of the spongiosa of the phalanges, characteristic of early sarcoid of bone. This was first pointed out by Jüngling.

with Still's disease was considered unusual. It was felt that if Still's disease were present the long duration of the illness should have brought out some of these signs.

The presence of so-called Clutton joints was suggested, although the Wassermann reaction of the patient's blood on three occasions and that of her mother gave negative results.

The results of the routine laboratory procedures offered no clue as to the nature of the disease process. The reaction to tuberculin was negative with a

dilution as low as 1:100 as well as with the dilutions used as a routine. The blood picture showed no significant deviation from the normal. The red blood cell count varied from 4,160,000 to 5,440,000 and the white blood cell count from

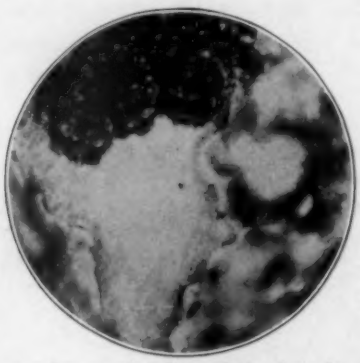

Fig. 4 (case 1).—An arthroscopic view of the interior of the right knee joint. Observe the clusters of sarcoid tissue hanging in the joint. The fat pads are also shown.

Fig. 5 (case 1).—A section of synovia from the right wrist, showing the formation of tubercles in the synovial and subsynovial layers.

6,100 to 10,000, with a percentage of neutrophils of about 65 and a lymphocyte range of from 22 to 47 per cent. Repeated examination of specimens of synovial fluid revealed a sterile fluid, with a varying admixture of red cells. Routine and

special (Corper's method) cultures of the fluid did not yield the tubercle bacillus or, for that matter, any other organism. An intensive attempt was made to demonstrate the tubercle bacillus after the microscopic demonstration of the disease, yet repeated inoculations of guinea-pigs and intraperitoneal and intratesticular injections into rabbits did not reveal the presence of the tubercle bacillus. The tracheobronchial glands, which were said by Frazier and Hu [3] to be the surest site for demonstration of the tubercle bacillus in animals inoculated with material from lesions of sarcoid, were not examined in these animals.

Fig. 6 (case 1).—A section of tissue removed from the right elbow, showing the tubercles penetrating between the muscle bundles.

On September 23 a tonsillectomy was done. On October 5 the removal of several teeth, the exploration of a small cavity in the olecranon process of the left elbow, which was demonstrable by roentgenogram, and an arthroscopic visualization of the right knee were carried out simultaneously with the patient under general anesthesia. The cavity in the olecranon process contained some granulation tissue, which yielded negative results on examination for a tuberculous process or for any specific disease. The significant observations on arthroscopic exam-

3. Frazier, C. N., and Hu, C. K.: Isolation of Treponema Pallidum from a Subcutaneous Sarcoid, Proc. Soc. Exper. Biol. & Med. **30**:898, 1933.

ination included peculiar grapelike clusters of reddish bodies attached to the synovia. The synovia was hypertrophic, and the cartilage of the joint was normal. A biopsy specimen was removed through a punch, and microscopic examination revealed nonspecific chronic synovitis; the specimen had evidently not been taken from one of the clusters. It was determined that an unusual form of synovitis existed, one which we had never before encountered. The patient's convalescence from these multiple procedures was uneventful.

On December 9 the left peroneal sheath was explored. The sheath was filled with a reddish clump of tissue similar to that seen in the knee joint through the arthroscope. The peroneal tendon was intact, but in its course through the masses of diseased tissue it was covered with a thin pannus. Microscopic examination of the excised synovia revealed numerous cellular tubercles, with giant cells, the nuclei of which sometimes numbered 15, numerous fibroblasts and numerous epithelioid cells. These tubercles were not caseous or necrotic and were clearly demarcated from the surrounding fat. No perivascular inflammation of note was seen. Dr. Henry Jaffe, pathologist of the Hospital for Joint Diseases, considered the disease to be sarcoid, a form of tuberculous involvement most frequently encountered in the skin as Boeck's sarcoid.

The unity of the disease process in the various tendon sheaths and joints was demonstrated repeatedly on microscopic examination of tissues removed at operation. The right knee was operated on on December 28, and the red, spongelike synovia was excised. The menisci were also removed. A small pannus of this abnormal tissue was present over one condyle of the femur, and this was removed with some difficulty. Convalescence was uneventful, and motion in the knee soon became complete. The left knee was opened on Feb. 8, 1933, and on incision of the synovia the red-purple masses popped out through the line of incision. A synovectomy was done. Unfortunately an infection of a hematoma in the quadriceps bursa developed, which was relieved by proper drainage. This, however, left the knee with a partial permanent limitation of motion, extension being full but flexion being permitted only to an angle of about 150 degrees. The masses in the right peroneal sheath were removed on April 26. After these procedures the patient was discharged.

She was admitted to the hospital again in the latter part of 1933, for further surgical work. On November 29 a partial posterior synovectomy of both elbow joints was done, with removal of the masses on the inner aspects of both elbows. These communicated definitely with the elbow joint proper. At the same time the masses within the tendon sheaths of the extensor carpi radialis brevis, the extensor carpi radialis longus and the extensor digitorum longus muscles were excised. Convalescence was uneventful, and on December 13 the last operation was done, at which time the masses of diseased sarcoid tissue were removed from the extensor digitorum longus and the peroneus tertius tendon and from the sheaths of the tibialis posterior and flexor digitorum longus muscles of the right foot. The sarcoid tissue was light pink and more fatty than in the other areas operated on. In the sheath of the extensor tendons one rice body was found; this was the only rice body noted at any of the operations.

The child was soon discharged and has since been seen periodically in the clinic. On her last examination, on April 30, 1935, there was moderate effusion within the right knee, with a partial recurrence of synovial thickening. The left knee showed no change. Slight recurrence was noted along the right peroneal sheath in its lower part. A new focus had developed along the sheath of the tendon of the left tibialis anterior muscle. Another focus had developed in the tendon

sheath on the dorsum of the left wrist. A recurrence was observed on the inner side of the left elbow. This proves that recurrence may take place despite the surgical removal of foci, although it has been established that the end-result of surgical intervention is good, since recurrence was not noted at most of the operative sites. That the disease is still active is manifested by the new foci.

The roentgenogram of all the joints showed no abnormalities save for the tiny cavitation in the ulna. This has increased slightly in size since the operation on the olecranon. On Nov. 21, 1933, the left femur showed a small gouged-out area on the anterior aspect of the internal condyle. The fingers and the toes from the time of the first admission showed a peculiar punctate atrophy the significance of which was not known. In the light of the later diagnosis of sarcoid the changes in the fingers and toes are typical of the early stage of sarcoid of the bone. A note on April 7, 1933, pointed out the conical shape of the fingers, the thickening being confined mainly to the soft tissues about the shafts of the distal two phalanges. The cavitation in the olecranon process and the subsequent cavitation in the internal condyle of the femur were evidences of sarcoid of the bone.

The microscopic observations point to the unity of the disease process in this patient. Most of the tubercles were located in the subsynovial layers and in the elbows; they invaded the triceps musculature, so that tubercles were seen between muscle bundles. The synovial villi at times showed the presence of these tubercles. The invasion of the muscle by the tubercles was noted only in the region of the elbow.

A careful study of the sections was made, and in none was caseation of the tubercles noted. Inoculation of guinea-pigs with tissues and fluid repeatedly failed to yield the tubercle bacillus. It was never possible to demonstrate the tubercle bacillus in properly stained sections of tissue.

We have not been able to find a record of a similar case in the literature. Faulkner [4] in 1931 reported the case of a woman of 49, with a loose, firm, small body in the knee joint. The microscopic picture was considered to be that of a sarcoid growth.

A good article for orientation on sarcoid is that of Goeckerman.[5] Sarcoid is best known as a cutaneous condition, especially of the Boeck type. Recently the visceral complications of sarcoid have been stressed, and Goeckerman noted that splenomegaly, infiltrations of the finer bronchioles of the lungs and fibrocystic changes in the bones may accompany sarcoid of the skin. It is striking that our patient presented no cutaneous evidence of sarcoid at any time. There was no splenomegaly, but a roentgenogram of the lungs showed a hilar adenopathy. The osseous changes of sarcoid have been stressed most by Jüngling, who termed the condition osteitis tuberculosa multiplex; more recently articles on sarcoid of bone have appeared by Bayer,[6] Van Alstine and

4. Faulkner, D. M.: Primary Synovial Membrane Tumors of Joints, Surg., Gynec. & Obst. **53**:189, 1931.

5. Goeckerman, W. H.: Sarcoids and Related Lesions: Report of Seventeen Cases; Review of Recent Literature, Arch. Dermat. & Syph. **18**:237 (Aug.) 1928.

6. Bayer, W.: Osteitis tuberculosa multiplex cystica mit Sarkoid und Hilusdrüsenbeteiligung, Jahrb. f. Kinderh. **138**:31, 1933.

Gowen,[7] Doub and Menagh,[8] Kirklin and Morton[9] and Sanes and Smith.[10]

There is a general opinion that sarcoid is due to the tubercle bacillus, despite the universal absence of a positive reaction to tuberculin, despite the inability to demonstrate the tubercle bacillus and despite the negative reactions in guinea-pigs. The organism is of low virulence, and the negative reaction to tuberculin is explained by the presence of a true anergy, a state of true immunity to the bacillus. The tubercle bacillus has been demonstrated occasionally in sarcoid lesions, especially in the early stages, before the true sarcoid appearance has developed. Kyrle[11] called sarcoid a foreign body reaction to the bacillus of tuberculosis and its disintegration products. Microscopically, the changes are not incompatible with those observed in tuberculosis, and in our case a study of each tubercle would have invariably led to a diagnosis of tuberculosis. Others believe in a more varied etiology, and the organism of syphilis especially has been held to be a causative factor. The pros and cons of this matter were well brought out by Goeckerman.

SYNOVITIS OF THE KNEE JOINT CHARACTERIZED BY BODIES RESEMBLING HASSALL CORPUSCLES IN THE SYNOVIA

CASE 2.—A woman, aged 37, twisted her right ankle and knee a year before she was admitted to the hospital. The right knee became painful and swollen. Aspiration was performed on the knee, and diathermy treatments were given. Subsidence of the pain took place in about a month, although a small amount of swelling remained. Nine months later severe pain and swelling of the knee again occurred, so that the patient was unable to bear weight on the right extremity. Six weeks before she was admitted to the hospital the knee again caused her to be incapacitated, so that further treatment was necessary. She walked with a knee limp on the right side, the knee being held at an angle of 170 degrees. Extension of the knee was possible to an angle of 170 degrees, and flexion only to an angle of 40 degrees. No abnormal movements were noted. Definite thickening of the synovial lining, especially in the quadriceps bursa, and enlargement of the fat pads were present. Slight tenderness existed along the line of the internal meniscus.

The roentgenogram of the knee showed no abnormalities. The only pertinent laboratory observation was a white blood cell count of 20,900. A diagnosis of

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7. Van Alstine, G. S., and Gowen, G. H.: Osteitis Tuberculosa Cystica Multiplex (Jüngling): Report of a Case Involving the Large Long Bones with Complete Proof of Its Tuberculous Etiology; A Review of the Literature, J. Bone & Joint Surg. **15**:193, 1933.

8. Doub, H. P., and Menagh, F. R.: Bone Lesions in Sarcoid; A Roentgen and Clinical Entity, Am. J. Roentgenol. **21**:149, 1929.

9. Kirklin, R. R., and Morton, S. A.: Roentgenologic Changes in Sarcoid and Related Lesions, Radiology **16**:328, 1931.

10. Sanes, S., and Smith, W. S.: Osteitis Tuberculosa Multiplex Cystica of Fibula and Tibia, J. A. M. A. **102**:1206 (April 14) 1934.

11. Kyrle, quoted by Goeckerman.[5]

hypertrophic synovitis of the knee joint was made, and arthroscopic visualization was advised to determine if the proper time for synovectomy was at hand. Examination was made on May 11, 1932, and a good view was had of the enlarged fat pads and tremendously hypertrophied but very inflamed synovia. It was therefore advised that synovectomy be postponed, in view of the acuteness of synovial inflammation. A cast was applied to the limb.

The patient wore the cast for a month and then received vaccine therapy for fifteen weeks, without much improvement. Pain developed in her right hand and right shoulder. She had also received thorough treatment privately. It was thought that the acute condition of the joint was passed and that synovectomy should be done. On October 12 the operation was performed. At operation the peculiar and interesting feature of the joint was the presence of whitish deposits

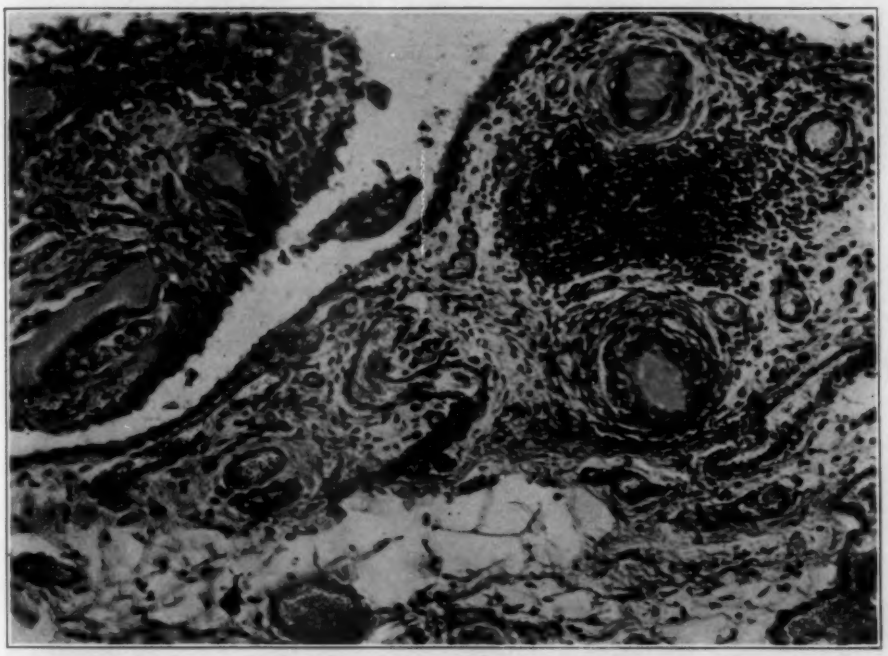

Fig. 7 (case 2).—A section of synovial membrane, showing the reactive bodies.

over the synovia, over the menisci and over some part of the cartilage, which resembled deposits of urate. The synovia was greatly hypertrophied otherwise, and the fat pads were much enlarged. The cartilage was in a good state of preservation. The synovia, fat pads and menisci were removed. On November 11 it was necessary to manipulate the knee to increase its range of motion. Otherwise, convalescence was uneventful. It should be noted that the white blood cell count just before the operation was still elevated to 16,800, with 67 per cent polymorphonuclears.

Chemically, the whitish deposit was definitely not urate but protein. The pathologic picture of the excised synovia was described as follows: There was slight hypertrophy of the synovial cells, with a slight tendency to the formation of villi. A general increase in vascularity existed, with lymphoid infiltration,

which at times formed little lymphoid nests. The peculiar and special character of the lesion lay in the presence of great numbers of small bodies just below the surface lining of the synovia, which at times resmbled Hassall corpuscles. The bodies possessed a central core of fibrillar, acellular material, with a peripheral, concentric mass of epithelioid cells and a more concentric proliferation of other surrounding tissue cells. Some of the bodies were surrounded by lymphocytic collections, and others were not. In some regions a healing process was taking place by fibrous, cellular proliferation of these bodies. Dr. Jaffe stated that such a histologic picture had not been previously observed in the laboratory.

The patient was admitted for the third time on Feb. 12, 1934, because of pain and a tired feeling in the knee on which the operation had been performed. Until recently she had been free from symptoms following the synovectomy. She also had pain in her right shoulder. Examination revealed some periarticular thickening of the knee. The patella was not as movable as it should have been. Some diffuse pain and tenderness were noted. Flexion of the knee was restricted to an angle of 90 degrees.

Arthroscopic visualization was therefore done again to determine what had happened to the joint after the synovectomy. The visualization of the joint was very good, and a mild reformation of the synovia, expressed in an irregular formation of villi most marked in the quadriceps pouch, was noted. The internal meniscus was not present, and regeneration had not taken place. Eosin introduced into the joint resulted in slight staining of the cartilage and uniform staining of the synovia. No indication for surgical intervention was seen. The patient received typhoid vaccine therapy for a short time. She was last seen in April 1935, still complaining of ache and weakness in the knee and of pain in her feet and shoulders. She is being treated as an arthritic patient.

MULTIPLE POLYPOID FIBROMATOSIS OF THE SYNOVIA OF THE KNEE JOINT

Multiple polypoid fibromatosis is an unusual involvement of the synovia of the knee joint, which clinically resembles nonspecific synovitis of the knee, with recurrent effusion. There is some resemblance to tuberculosis, and that condition has to be considered in the differential diagnosis. It is a true tumor of the knee, and case 3 was the first one of this type that we have encountered clinically. Dr. Jacobson told us that several specimens of synovia exhibiting such a tumorous change have passed through the laboratory.

CASE 3.—The patient was a Negress, aged 31, who was first seen in the dispensary in 1931. At that time she presented an effusion within the left knee joint, which had developed without trauma. The fluid was aspirated, and all efforts to prove the joint to be tuberculous were without avail. The patient did not return, nor did she seek medical attention elsewhere. The swelling of the knee had never completely subsided, and it was definitely worse during the menstrual periods. Pain had been present, especially on weight bearing. There was no history of locking. A week before her second visit to the clinic the swelling of the knee became much worse. Dull ache was associated with it.

The left knee showed marked effusion. The synovia was markedly thickened and could be felt almost as a distinct layer. Small discrete masses could be felt, which were attached to the synovia, especially one the size of a small almond

which was attached near the region of the internal meniscus. Several smaller masses were felt in the outer part of the quadriceps bursa. Tenderness was present over several synovial points. Flexion was limited to an angle of 90 degrees, and extension exceeded the normal limit of 180 degrees by 5 degrees. The right knee showed similar hyperextension.

The Wassermann reaction of the blood was negative. The reaction to the complement-fixation test for gonnorrhea was 3 plus. The reaction to the tuberculin test was negative with all diagnostic dilutions. Roentgenograms showed the absence of a joint body, and the suprapatellar pouch was divided into two compartments. A sample of synovial fluid did not show organisms, and a culture was sterile. Some lymphocytes, polymorphonuclear cells and red cells were noted in a smear.

Repeated aspiration of the knee was done before arthroscopic examination. Good visualization was had of the knee in the extended position. Visualization was not possible with the knee in the flexed position because of extensive synovial hypertrophy. Tremendous masses of fat were seen, which were irregularly placed but were greatest in the region of the fat pads. The fat pads were somewhat enlarged and were fuzzy on their inner surface. The quadriceps pouch was short and showed a tremendous formation of villi. The patella seemed normal. This extreme degree of synovial overgrowth within the joint seemed to justify a diagnosis of lipoma arborescens. Several specimens were taken for biopsy, since such an overgrowth of the synovia is occasionally caused by tuberculosis adjacent to the synovia. The pathologist was of the opinion that the fatty masses removed for biopsy had come from para-articular tissues and not from the joint itself. The patient sustained some irritation from the arthroscopic procedure, as indicated by increased effusion within the knee, local heat over the joint, and a reaction of the temperature, which, going to 102 F. on the second day, vacillated between 99 and 101 F. for the third, fourth and fifth day postoperatively.

Arthrotomy was done two weeks later, on Feb. 28, 1934, and the arthroscopic observations were verified in their essentials. Synovectomy was done, and the fat pads and both menisci were removed. The menisci were degenerated, and a pannus was present at the edges. The postoperative course was not eventful.

The pathologic observations were most interesting. Grossly, the specimen consisted of the quadriceps pouch, the two menisci and two other pieces of synovia removed from the knee. All the pieces had undergone a more or less uniform pathologic transformation. The essential change was the appearance of smaller or larger pedunculated, polyp-like masses attached to the synovial lining by short, thin stalks. The polyps were in a sense growths or outgrowths of the tissue below the lining of the synovial membrane. Aside from the striking polypoid appearance, the synovia bore the evidence everywhere of abnormal change in the same direction. In some places, there were fine, delicate villi. In other places, shining through the synovial membrane, there were tiny, whitish but flat miliary nodules. At one point in the subsynovial layer of the pouch a whitish, cartilaginous mass about 1 cm. in diameter was seen. This mass was partly discolored by brown pigment. In fact, all of the resected synovia was considerably discolored by hemosiderin pigment, which may possibly be associated with the innate character of the lesion. Fibrin was noted in some areas.

Microscopically, numerous large pedunculated outgrowths of the joint capsule lined by synovia were observed. The dominant cells were those with a small oval nucleus and with indistinct cytoplasm. These were for the most part massed closely together in enormous numbers. A striking feature was the development in more than a few places of intercellular matter, which was probably hyalin,

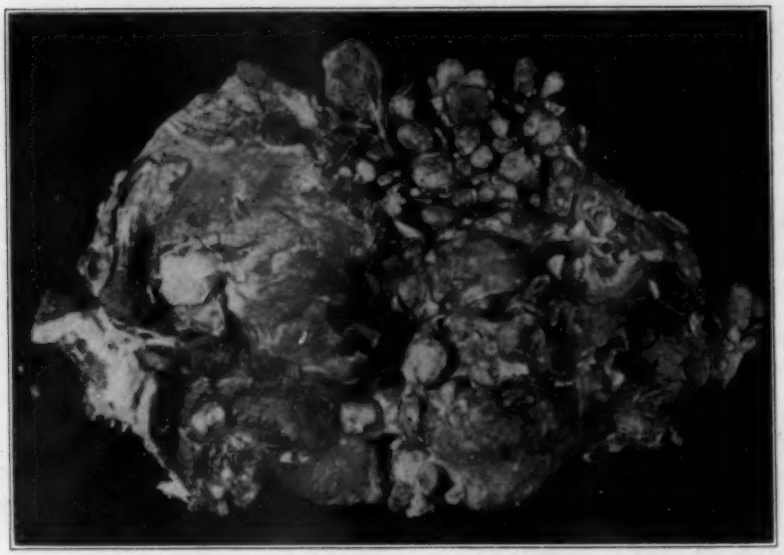

Fig. 8 (case 3).—A photograph of excised synovial membrane and fat pads. Note especially the numerous pedunculated, polyp-like masses attached to the hypertrophic synovia.

Fig. 9 (case 3).—A section of synovial membrane, showing villous hypertrophy, as well as a large, pedunculated nodule.

although suggestions of osteoid were not lacking. A moderate number of epulis-like giant cells, lymphocytes and fat-laden phagocytes were noted.[12]

A diagnosis of multiple, polypoid, hyalinizing, connective tissue outgrowths of synovial membrane (fibroma) was made.

When discharged several weeks after the operation, the patient was able to flex her knee to 90 degrees. There was still some swelling of the knee. In August 1934 she had no symptoms. There is still atrophy of the quadriceps muscle. Crepitation is present on motion, and flexion is limited to 85 degrees. Extension is full.

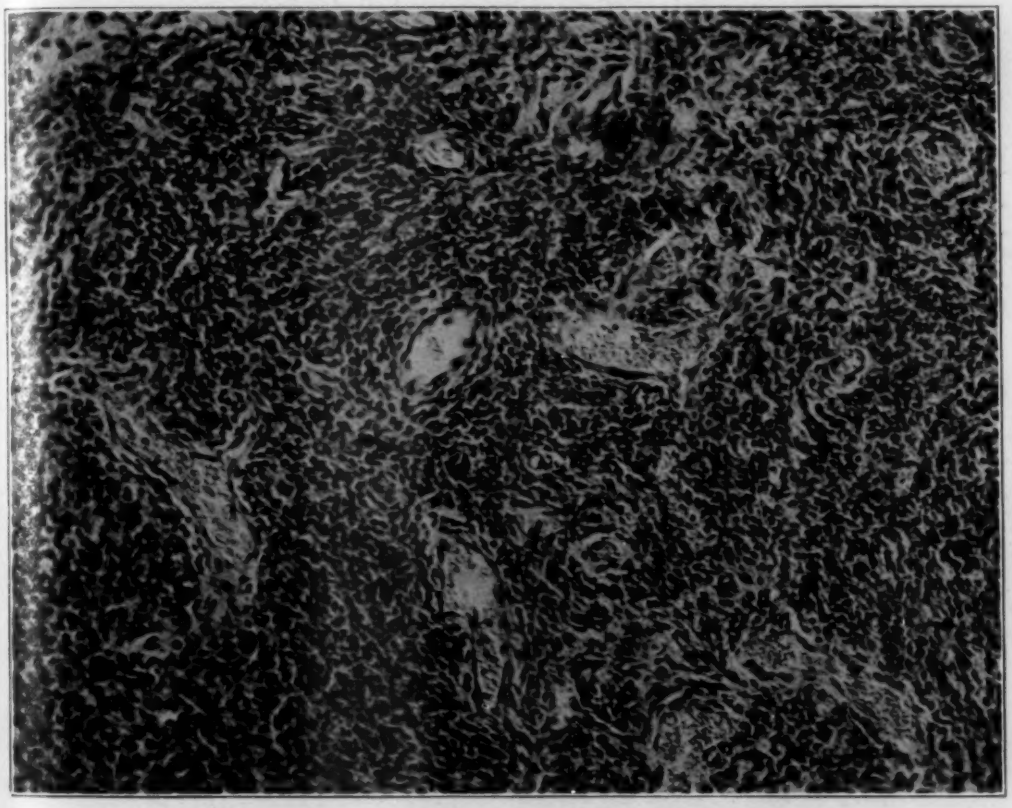

Fig. 10 (case 3).—A high power magnification of a tumor nodule.

CHRONIC NONSPECIFIC SYNOVITIS OF THE KNEE JOINT IN YOUNG PATIENTS

There is a typical disease entity of the knee joint in young patients which is perhaps best expressed by the foregoing title. The involve-

12. The report also read: "Groups of very large, pale reticular cells, with small more or less centrally placed round nuclei, are seen. They apparently represent the intracellular accumulation of lipoid."

ment is usually bilateral and is characterized by the presence of repeated effusions within the knee joint, with thickening of the synovial lining. Aspiration may diminish the effusion temporarily, but there is a marked tendency for rapid recurrence. Only the knee joint is involved, and no definite cause can be invoked. Trauma is apparently coincidental. The reaction to tuberculin is negative, and no signs of syphilis are present. The Wassermann reaction of the blood and of fluid from the joint is negative. These cases form a baffling group, not so much from the standpoint of therapy as from the standpoint of etiology. Possibly, some occult focus of infection exists in the body. It is always necessary to rule out a specific lesion, although the clinical behavior of the knee joints in these cases resembles that of syphilis more than that of tuberculosis. Sundt [13] and Gill and Orr [14] described this type of case accurately.

Two cases of this particular disease group are included in this report, one in a youth of 18 and the other in a girl of 12 years. Each case is interesting enough for separate description. The value of arthroscopy lies in the clear examination of the joint and the possible therapeutic effect of lavage. It is possible to rule out tuberculosis by examination of the joint and by removal of a specimen for biopsy. Other suspected lesions are easily ruled out, and the disease process is seen to be confined to the synovia.

Case 4.—Alice S., a girl aged 12 years, fell and hurt her left knee while skating five years before she was admitted to the hospital. The knee was said to have been swollen, tender and discolored, but the condition cleared up after two weeks. The patient had scarlet fever in 1932, but no joints were involved as a sequel of the disease. Her left knee became swollen in October 1933, again after skating, although this time she did not suffer a fall. The left knee became progressively swollen, and at times it seemed hot and tender to touch. The pain was located on the inner side of the knee, was worse in the morning and was relieved by walking. Some limitation of motion was noted due to pain. No general symptoms, except possible excessive sweating in her sleep, had been noted. Another fall on the knee was noted, although the date of its occurrence was not remembered.

The child was well developed and seemed to be in perfect health. General examination gave entirely negative results. The left knee showed a large effusion, with some synovial thickening. Motion was free. The right knee which had always been free from disability, according to the patient, showed a smaller effusion. Its motion too was free. No tenderness existed in either joint. Serologic

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13. Sundt, H.: The Diagnosis and Frequency of Tuberculous Disease of the Knee, J. Bone & Joint Surg. **13**:740, 1931; Ueber Gonitis incertae causae insbesondere Synovitis chronica "simplex," unter besonderer Berücksicktigung des "Rheumatismus tuberculosus-Poncet" und Fällen von Synovitis mit ausgesprochener fleckiger Atrophie, Acta orthop. Scandinav. **3**:97, 1932.

14. Gill, A. B., and Orr, T. E.: Chronic Synovitis of the Knee with Persistent or Recurring Effusion and of Undetermined Etiology, J. Bone & Joint Surg. **16**: 159, 1934.

tests for syphilis and gonorrhea gave negative results. The roentgenogram showed an essentially normal picture.

Examination of synovial fluid showed a moderate number of lymphocytes and polymorphonuclear cells. No bacteria were seen. The Wassermann reaction of fluid from the joint was negative. Culture of the fluid and inoculations of guinea-pigs yielded negative results for tuberculosis. The sedimentation time of the blood was at a high normal level.

Arthroscopic observation of the left knee was done on Dec. 7, 1933. Visualization was perfect. An interesting lesion of the synovia was noted. Fringing each border of the patella were large, oblong villi, which presented an outer third of clear yellow and an inner, clearly demarcated two thirds of delicate rose-pink. The infrapatellar fat pads were slightly enlarged. Some synovial hypertrophy existed in the quadriceps bursa. At the base of the intercondyloid fossa and just anterior to it were a large number of small, petal-like villous structures, with the same type of coloring noted in the villi about the patella. These villi were fringed like coral, and their superior edges were thus slightly serrated. The joint cartilage was normal. The internal meniscus was well visualized and was normal. No specimen was taken for biopsy. Convalescence was uneventful, and the effusion within the knee did not return.

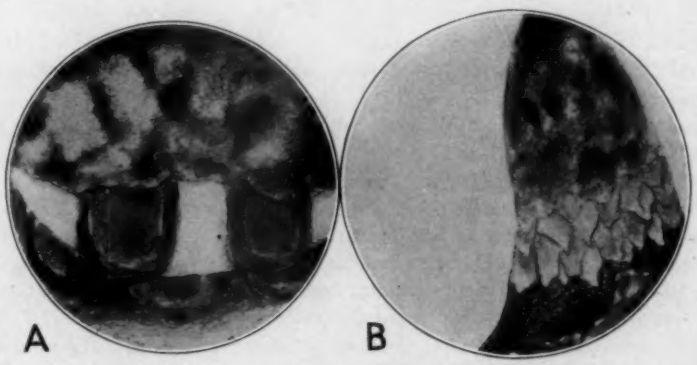

Fig. 11 (case 4).—*A* is an arthroscopic view of the left knee joint, showing large, oblong-shaped villi fringing the patellar border. The outer third of each villus is a clear yellow and is clearly demarcated from the inner two-thirds which is rose pink. The cartilage of the under surface of the patella is normal. *B* shows a large number of coral-like villi, serrated at their tips, seen at the base of the intercondyloid fossa. They are colored as the oblong villi about the patella.

Arthroscopic examination of the right knee was done two weeks later. At this time no effusion was present in the left knee. The right knee joint was clearly visualized. The observations were in many ways similar to those seen in the opposite knee. The synovial villi about the patella were diaphanous and showed only a suggestion of the unusual coloring noted in the other knee. The oblong shape of the villi was noted. On the medial aspect of the joint there was a large collection of thickened and roughly oblong villi, so that visualization of the internal meniscus was incomplete. The suggestion of the unusual coloring was noted here too. The base of the intercondyloid fossa did not show the synovial villi noted in the left knee. Otherwise, the observations on the rest of the joint corresponded with those on the left knee. The biopsy specimen showed the presence of non-specific chronic synovitis.

The effusion within this knee disappeared rather quickly, so that when the patient was discharged, on December 27, the knees seemed normal. On Feb. 14, 1934, a note made in the clinic testified to the objective normality of her knees. On April 11, despite a fall on the knees three weeks before, they were still without further pathologic changes.

It cannot be said that this patient is cured, and only time will tell if recurrence of the effusion takes place. The effectiveness of therapeutic lavage is demonstrated here. The history of trauma is confined to the left knee, yet both knees were involved. We believe that the trauma was coincidental.

Case 5.—Jacob C., a youth aged 18, was first admitted to the hospital in 1928, because of questionable tuberculosis of the tendon sheath of the tibialis posterior tendon. Recovery was uneventful, and the tuberculous nature of the swelling about the tendon was never demonstrated. The knees were normal at this time. The patient's present illness, swelling of both knees, came on about three weeks before he was admitted to the hospital on Dec. 18, 1933. On waking in the morning he noted that his knees had become swollen. He had had no previous illness recently, and no injury was recalled. The swelling of the left knee was marked, whereas the right knee was only slightly swollen. No pain and no tenderness were noted. He had lost about 4 pounds (2 Kg.) since the onset of his illness. About 150 cc. of fluid was removed from the left knee, but the effusion quickly returned.

The patient was tall and thin and walked with a definite knee limp. The left knee was full of fluid. Extension was possible to an angle of 165 degrees, and flexion to an angle of 75 degrees. The right knee on standing was held in a position of 175 degrees of extension, and extension was not possible beyond this point. Flexion of the right knee was possible to 55 degrees. No redness, heat or spasm was noted about either knee. No swelling of the sheath of the tibialis posterior muscle was noted. The patient's general health was good, and examination of the other systems of the body gave negative results.

Roentgenograms of both knees failed to show abnormalities. The Wassermann reaction of the blood was negative. The reaction to tuberculin was also negative. The sedimentation time of the blood was slightly increased. Repeated examination of the synovial fluid gave negative results culturally, and inoculations in guinea-pigs gave no evidence of tuberculosis. The Wassermann reaction of the fluid of the joint also gave negative results.

In the attempt to prepare the left knee for arthroscopic examination frequent aspirations of the knee were done. It was never possible, however, to reduce the effusion materially because of the rapid accumulation of fluid within it. It was finally decided to perform an arthroscopic examination of the right knee on the basis of the similarity of the pathologic process in each knee and to give the left knee lavage for any therapeutic effect it might have. The visualization of the right knee was excellent and demonstrated the synovial hypertrophy quite well. A small area of erosion on the under-surface of the patella was well brought out with eosin. Lavage to the left knee was given with over 500 cc. of physiologic solution of sodium chloride. A biopsy specimen from the right knee indicated the presence of subacute synovitis.

Lavage of the left knee had no effect on the magnitude of the effusion, for it was necessary to aspirate the knee frequently. The right knee showed about the same amount of fluid as was present before arthroscopic visualization. Hence,

three weeks later a synovectomy of the left knee was done. Pathologic examination of the excised tissue revealed nonspecific chronic synovitis. An interesting sympathy was noted in the subsidence of the effusion of the right knee after the synovectomy of the left knee. Symptoms in the right knee also disappeared. The patient was discharged on March 6, 1934.

It was necessary to admit the patient again in the latter part of October because of the recurrence and persistence of fluid in the right knee. A synovectomy was done on this knee.

An interesting point in the history during the interval related to a transient swelling of one sternoclavicular joint in June. This swelling subsided promptly without treatment. No evident focus of disease has been found. The patient has been followed up regularly in the outpatient-department. Motion in both knees is good, albeit with slight limitation in flexion. Fluid has recently reappeared in the left knee (April 1935) controlled by a simple compression bandage. One disturbing feature has developed, epididymitis of unspecific origin. The patient gets about well and wishes to go to a camp of the Citizens' Conservation Corps.

It is important to differentiate the condition in this group of cases from specific disease of the knee joint and from those nonspecific diseases of the knee joint which simulate a specific disease and are accompanied by local inflammation or the definite signs of the disability of the knee after trauma. This group is limited to a small percentage of cases of involvement of the knee. A long follow-up in this type of case may ultimately disclose involvement of other joints, the usual nature of the arthritic process being delayed or masked by the predominant disease of the knees.

OSTEOCHONDRITIS DISSECANS

Case 6 was noteworthy because of its unusual features and because of certain points of view regarding etiology and treatment which it brought up. Although Conway,[15] Fairbank,[16] Balensweig,[17] Filippi,[18] Delchef,[19] Heine,[20] and Niessen[21] described cases of osteochondritis dissecans of the external condyle of the femur, in none was there a location of the disease process similar to that in this case. Hellström[22]

15. Conway, F. M.: Osteochondritis Dissecans, Ann. Surg. **99**:410 (March) 1934.

16. Fairbank, H. A. T.: Osteochondritis Dissecans, Brit. J. Surg. **21**:67, 1933.

17. Balensweig, I.: Osteochondritis Dissecans of the Knee, J. Bone & Joint Surg. **7**:465, 1925.

18. Filippi, G.: Contributo allo studio della osteocondrite dissecante (König), Chir. d. org. di movimento **16**:35, 1931.

19. Delchef, quoted by Fairbank.[16]

20. Heine, J.: Beitrag zur Pathogenese der Osteochondritis Dissecans, Deutsche Ztschr. f. Chir. **206**:119, 1927.

21. Niessen, H.: Ueber symmetrische Osteochondritis dissecans der lateralen Femurcondylen, Beitr. z. klin. Chir. **143**:159, 1928.

22. Hellström, John: A Few Experiences in Reference to Osteochondritis Dissecans, Acta chir. Scandinav. **74**:427, 1934.

described sixty-nine cases of osteochondritis dissecans, in three of which the site was in the external condyle.

CASE 6.—R. H., a Negro aged 17, was first seen on Aug. 17, 1933, because of a gradual swelling of his left knee, which had been manifest for six weeks and which had not been induced by any known trauma. No pain had been complained of. Frequently the patient felt that something was slipping on the inner side of his knee, but no real locking had taken place. Simple straightening of the leg removed this sensation. Examination revealed moderate effusion within the left knee joint. On the inner side, medial to the patella and just above the line of the internal meniscus, a pea-sized body was felt, which could be slipped in and out. This manipulation was painful. Motions of the knee were normal. After the aspiration of 75 cc. of a yellow-brown, syrupy fluid, this body was more easily palpated.

In view of the history and physical findings, a presumptive diagnosis of osteochondritis dissecans was made. Yet the presence of the body near the internal meniscus suggested also the much rarer possibility of a tumor, such as a fibroma or a xanthoma, arising from the internal meniscus.

The patient was admitted to the Hospital for Joint Diseases on August 21. The joint body had slipped into the joint space the day before admission; in fact, it could no longer be palpated. There was now no effusion. On motion of the knee the synovia gave forth rustling and crackling sounds, indicative of the presence of synovitis. No tenderness was present over the region of the internal meniscus or elsewhere within the joint.

A roentgenogram of the knee gave no indication of foreign body or other intrinsic disease of the joint. The systematic examination of the synovial fluid also gave essentially negative results; this examination included a Wassermann test and culture of the fluid and inoculation of a guinea-pig. The smear of the fluid showed occasional red blood cells and white blood cells. The Wassermann reaction of the blood was also negative.

Arthroscopic visualization with the patient under general anesthesia and immediate arthrotomy were done on August 23. Unfortunately, because of a faulty electrical connection, it was necessary to interrupt the arthroscopic examination after a few minutes' examination. Small, continuous shocks were felt by the operator, and the patient experienced one involuntary but painless stimulation of his knee into extension. Yet, even in the brief time of examination, it was possible to note through the usual external puncture that the cartilage of the internal condyle of the femur was intact, that the internal meniscus was normal, that the patella was normal and, finally, that the quadriceps bursa was the seat of a definite and moderately advanced chronic synovitis.

Before the joint was opened, 20 cc. of a 0.05 per cent aqueous solution of eosin was injected into the joint, and 15 cc. of this was obtained by immediate aspiration. The usual median parapatellar incision was made. A striking and unusual picture of disease was now seen. The synovia of the bursa was moderately inflamed and fuzzy and presented a definite villous synovitis of from mild to moderate degree. The synovia was stained a light pink by the dye. The infrapatellar fat pads were slightly enlarged but not injected. The ligamentum mucosum was a reddish, somewhat inflamed band of tissue. Near the junction of the midportion and the upper portion of the external condyle a square erosion in cartilage about ¾ inch (2 cm.) square was noted, demarcated from the surrounding normal white cartilage by a light brown border of loose cartilage about

1/16 inch (0.15 cm.) wide. Within this erosion, the base of which was not bone but an irregularly papillated cartilage, were two free fragments of normal-appearing cartilage of equal size. The dye had stained the eroded area a light pink, whereas the normal cartilage had not been stained. The cartilage of the internal condyle was normal, and no area of dissection was noted in it. In addition, a red fibrous membrane of heavy pannus spread out from the anterior cruciate ligament and attached itself along the free border of the internal meniscus along its entire length. Beneath this, and over the internal condyle of the tibia, another square piece of white cartilage, about ¾ inch square, was noted. Another body, the size of half a pea, was observed between the outer part of the external condyle of the femur and the lateral synovial expansion. Both menisci were intact.

Both free bodies were easily removed from the ulcer in the cartilage in the external condyle. The loose brownish border was excised at its junction with normal cartilage. The small body laterally was also removed. A slit was made in the reddish membrane, and the body over the internal condyle of the tibia was

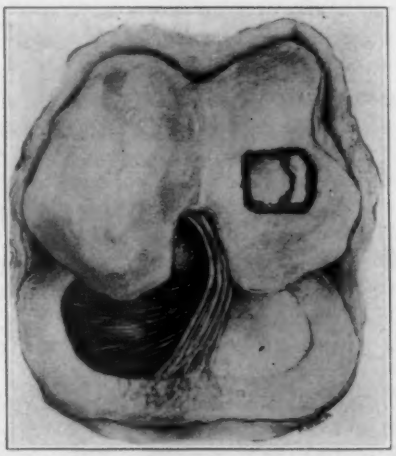

Fig. 12 (case 6).—A drawing of the opened knee, illustrating the pathologic changes noted in the knee. The ulcer of the external condyle of the femur, with its two pieces of cartilage, is evident. The reddish, fibrous pannus extends from the anterior cruciate ligament to the internal meniscus, attaching along the entire free border. It covers over another loose piece of cartilage the origin of which is probably from the internal condyle of the tibia.

taken out. Its origin, presumably from the internal condyle, could not, however, be verified by direct inspection, since this would have necessitated a close dissection of the pannus, a thing which was not considered advisable. A synovectomy was done by which the synovia was removed from the bursa under the quadriceps tendon and its lateral expansions, and the inner fat pad was removed. The altered ligamentum mucosum was not touched.

The wound was closed in layers, and a cast was applied from the toes to the groin. Convalescence was uneventful, the highest peak of temperature being 101.4 F. By the twelfth day there was no further rise from the normal. The cast was removed soon afterward, and active motion of the knee was begun. This

proceeded satisfactorily, and it was not necessary to manipulate the knee at any time.

On September 13 an unsuccessful attempt was made to visualize the lateral vessels of the knee by the closed method of arteriography, as advocated by Leriche. A cuff was tied just below the knee to prevent the passage of the contrast medium (thorium dioxide) below the leg. The femoral artery was felt slipping away from the needle, and it was not possible to enter it. Instead, a vein was entered, and some of the fluid refluxed into the abdominal veins. Roentgenograms made twelve seconds and four minutes, respectively, after the injection showed no abnormality. It was intended to determine if an embolic block existed in the lateral vessels of the knee.

The fact that the erosion in the cartilage did not reach to the bone apparently speaks for a factor acting locally, such as an unnoted trauma acting on the external condyle of the femur when the patella abutted against it in flexion and external rotation of the knee. In some persons the knees in the flexed position expose a large part of the femoral condyles, and even without patellar contact it may be possible to exert direct trauma to the cartilage. Yet it is well known that it is most difficult to fracture normal cartilage, even with a strong blow.

The irritation of the joint bodies created a state of chronic synovitis. This synovitis does not necessarily disappear after the removal of the joint bodies, and occasionally it may be advisable to remove the diseased synovia by synovectomy. The remaining portion of the ulcer in the cartilage is said to fill in with fibrous tissue or fibrocartilage, so that ultimately only a small depression is noted at the site of the disease. It is of course well known that cartilage does not heal over well. Clinically, this is also true, and these joints must always be considered as subnormal, with the ever present possibility of the development of arthritis sooner or later. In experimental animals a defect in the cartilage often leads to widespread arthritis. This was proved by the work of Key,[23] and Burman and Sutro.[24] A long follow-up of patients with this condition should indicate a greater frequency of such arthritic changes.

The patient was discharged on September 14. On October 21 he stated that he was entirely cured. A belated roentgenogram of the spleen and liver made to determine the presence of thorium dioxide showed no abnormality. On October 24 it was noted that extension of the knee was full and that flexion was possible to 95 degrees. There was no swelling and no crepitation on motion. Good power was present in the quadriceps muscle. On December 26 the patient was able to walk an unlimited distance and was without symptoms. Extension was full, and flexion was possible to 85 degrees. The patient was last seen on April 20, 1934. His knee showed a slight limitation in extreme flexion but was normal otherwise. He could play basket-ball, run or walk without disability. He considered his knee to be entirely normal.

Microscopic examination of the excised cartilage and synovia did not help us from an etiologic standpoint. The cartilage showed hypercellularity of the superficial layers and some localized thickening of the perichondrium. The synovia showed some villous hypertrophy and was the seat of chronic synovitis.

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23. Key, J. A.: Experimental Arthritis: The Changes in Joints Produced by Creating Defects in the Articular Cartilage, J. Bone & Joint Surg. **13**:725, 1931.

24. Burman, M. S., and Sutro, C. J.: Staining of Cartilage: Gross Staining by Intra-Articular Injection of Dyes in Animals, Arch. Surg. **27**:801 (Oct.) 1933.

SOLITARY LIPOMA OF THE KNEE JOINT

We have had the opportunity of observing two cases of solitary intra-articular lipoma of the knee joint. This is an uncommon tumor of the knee joint, the primary clinical importance of which lies in the fact that it acts as a mechanical block to motion, producing repeated temporary locking. It is our opinion that the solitary lipoma arises in the presence of other diseases of the synovia and that it is such synovial irritation which rarely allows a synovial villus to proliferate so that a true lipoma is formed. This corresponds in general with the ideas of Otterbeck [25] and Filter [26] and is in accordance with clinical observations. We do not take much stock in the theory of König that a solitary lipoma arises through rupture of the synovial membrane, with intra-articular protrusion of subcapsular fat.

According to Stieda,[27] only twelve cases had been described up to 1896, and since then less than an equal number have been reported in the literature. Geschickter,[28] in a review of cases of lipoma and lipoid tumors, noted lipoma of a joint in seven of a group of seventy cases of lipoma of the breast and viscera. Occasionally, and in earlier reports, some confusion of a solitary lipoma with enlargement of the infrapatellar fat pads (Hoffa's disease) is noted.

While a history of direct trauma may be obtained, such as a direct fall on the knee, it is most likely that the trauma came about either coincidentally or as a result of the interposition of the tumor between the bony surfaces of the joint. This certainly was true in our second case and was definitely proved in the case of Herhold,[29] in which the patient, a cannoneer, had noted a tumor before the gross trauma occurred which brought him to consult the physician. The tumor occurs at all ages and in both sexes. Pain is variable. It may not be present or it may be severe, especially at the moment of temporary locking. The swelling of the knee may be due to effusion, to the presence of the tumor, to the presence of coexisting synovial disease or to a combination of these factors.

The lipoma may rest dormant, or its gradual growth may interfere with motion. Its size varies, a maximum of 3½ inches (9 cm.) having been reached in the case of Hunt.[30] The tumor is well outlined and

25. Otterbeck, W.: Freies Lipom im Kniegelenk, Inaug. dissert., Bonn, J. Bach, 1892.

26. Filter, M.: Ueber solitäre Lipoma des Kniegelenks und ihre Ursachen, Inaug. dissert., Greifswald, J. Abel, 1890.

27. Stieda, H.: Ueber das Lipoma arborescens des Kniegelenks und seine Bedeutung zu chronischen Gelenkaffektionen, Beitr. z. klin. Chir. **16**:285, 1896.

28. Geschickter, C. F.: Lipoid Tumors, Am. J. Cancer **21**:617, 1934.

29. Herhold: Zur Frage des Lipoma Genu, Arch. f. klin. Chir. **52**:705, 1896.

30. Hunt, E. H.: A Case of Lipoma of the Knee, St. Barth. Hosp. J. **9**:121, 1902.

may have a soft, thick or even semifluctuant feel, so that its growth beneath the patella in the case of Nodet [31] gave the impression of pseudoballottement. The tumor may slip in and out of the joint on motion, or it may be forced manually into the joint. The location varies, though it is often located near the alar ligaments. It may arise from the quadriceps bursa or it may be felt in the popliteal fossa. In Herhold's [29] case, the tumor was attached by a pedicle to the anterior cruciate ligament. Motion of the knee joint is usually not limited, but in Auvray's [32] case flexion was limited to 90 degrees both actively and passively.

While the presence of the tumor is confirmed at operation, the small incision used for its removal does not allow an adequate survey of the entire joint, so that many authors, while mentioning synovial irritation as a possible etiologic factor, made no note of the state of the synovia. In Züllig's [33] case a lipoma was removed at one point within the joint, but it recurred at another point.

Mixed tumor may occur. The tumor may be both intra-articular and para-articular, as in Campbell's [34] case. However, in this case the diagnosis was roentgenologic, and the supposed masses of fat, which cast a dense shadow on the roentgenogram, were not verified by opening the joint. Malignant change to liposarcoma took place in Auvray's [32] case, and Blagovïeshtshenski [35] was cited by Auvray as having described a case of liposarcoma of the knee joint. We do not know whether the growth was a solitary tumor.

The tumor in the case of Simon,[36] which is considered to be the first case described, was apparently not a lipoma but a fibroma. Volkmann,[37] then, is to be given priority for the first description of solitary lipoma of the knee. Filter,[26] Otterbeck,[25] Annandale,[38] Barwell,[39]

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31. Nodet, V.: Le faux choc rotulien dans un cas de lipôme du genou, Province méd., Lyon **10**:492, 1896.

32. Auvray: Lipo-sarcome primitif intraarticulaire du genou, Bull. et mém. Soc. de chir. de Paris **46**:534, 1920.

33. Züllig, J.: Tumoren der Kniegelenkkapsel, Cor.-Bl. f. schweiz. Aerzte **47**: 1368, 1917.

34. Campbell, D.: Intra- und extraartikulare Lipome des Kniegelenks, Fortschr, a. d. Geb. d. Röntgenstrahlen **37**:852, 1928.

35. Blagovïeshtshenski, D. A.: Dendroid Lipomatous Tumor of the Knee Joint, Khirurg. Arkh. Velyaminova **27**:653, 1911.

36. Simon, G.: Exstirpation einer sehr grossen, mit dickem Stiele, angewachsenen Kniegelenkmaus mit glücklichem Erfolge, Arch. f. klin. Chir. **6**:573, 1865.

37. Volkmann, quoted by Stieda.[24]

38. Annandale, T.: An Address on Internal Derangements of the Knee Joint and Their Treatment by Operation, Tr. Med.-Chir. Soc. Edinburgh **6**:78, 1886-1887.

39. Barwell, quoted by Annandale.[38]

Weir,[40] Stieda, [27] Fisher,[41] Wagner,[42] Lauenstein,[43] and Bland-Sutton [44] also described cases.

The exact diagnosis is sometimes difficult, even if solitary lipoma is thought of. An exact preoperative diagnosis may be difficult, since a fibroma, a chondroma, a pedicled sarcoma, a xanthoma, an angioma, a pedicled sarcoid, a meniscal cyst or tumor, a synovial cyst or an ordinary rounded joint body may produce a similar picture of disease. The consistency of the tumor, the character of the synovial fluid, which may be bloody with angioma, xanthoma or sarcoma, may help in the diagnosis. A high value for cholesterol in the blood is not uncommon with xanthoma, and a determination of the cholesterol content of the blood should be carried out in every doubtful case. The presence of hypercholesteremia may be a guide to an exact diagnosis. The roentgenogram usually fails to show an abnormality, although it is possible that the fat may cast a shadow, especially if calcium is deposited within it.

Treatment is surgical and consists in the removal of the tumor. The tumor should be transfixed by two needles just before the synovia is entered; otherwise, it may be difficult to locate it properly. The ultimate fate of the joint rests on the coincident pathologic process in the joint.

CASE 7.—Raoul D., a man aged 24, a Santo Domingan who had recently come to New York, was seen first on May 1, 1933, because of pain in his feet. While in Santo Domingo, one and a half years previously, he had gonorrhea, for which he was treated rather perfunctorily. He was in bed for three months because of the development of acute gonorrheal arthritis of the left knee. No treatment was given, and the disease subsided spontaneously. Motion in the knee was restored by active exercises instituted on the patient's own initiative. The right knee was not involved, according to his statement. He had received intravenous injections, apparently for syphilis, at another hospital. The gonorrheal urethritis was cured, but since getting out of bed the patient had had pain in both knees and both feet. He was given orthopedic shoes by a chiropodist, and two and a half weeks later he first noted a small tumor on the outer side of his right knee. This slipped in and out on motion. There was no trauma at any time to the knee.

A tumor the size of a dime was noted on the external aspect of the right knee joint. The tumor was not painful on pressure and had the consistency of thickened fatty tissue. It slipped in and out of the joint between the angle of 180 and that of 170 degrees of motion and caused neither pain nor temporary locking in its

40. Weir, R. F.: On Fatty and Sarcomatous Tumors of the Knee Joint, M. Rec. **29**:725, 1886.

41. Fisher, A. G. T.: Internal Derangements of the Knee Joint, New York, The Macmillan Company, 1924.

42. Wagner, A.: Ueber Lipome des Kniegelenks, Inaug. dissert., Königshütte, R. Giebler, 1887; quoted by Herhold [29] ad Stieda.[24]

43. Lauenstein: Centralbl. f. chir. **11**:1, 1884; quoted by Stieda [24] and Weir.[40]

44. Bland-Sutton, J., quoted by Herhold.[29]

excursion. It was located at the lower pole of the patella. It was thought that the tumor might come from the external fat pad, although it seemed to be placed too high. Slight atrophy of the quadriceps muscle was present. Motions of the knee were complete but crepitant. The left knee also showed crepitation on motion.

Both feet, especially the right, were painful, and without shoes, the patient walked with a perceptible limp. There was marked atrophy of the muscles of both calves, so that the patient could not stand on tiptoe well. Tenderness was present over the tuberosity of the fifth metatarsal bone of the right foot, just behind the tuberosity and over the entire shaft of the fifth metatarsal bone. Tenderness was also present over the external aspect of the ankle and over the medial aspect of the heel. The metatarsal heads were also tender. The peroneal tendons were slightly taut. Inversion of the foot was limited, and subastragaloid motion was definitely painful and limited. Forced inversion increased the pain, especially in the region just behind the tuberosity of the fifth metatarsal bone. Some swelling was present around the ankle joint, around the subastragaloid joint and in the region of the ball of the foot. Dorsiflexion of the foot was possible to an angle of about 90 degrees. Mild pronation of both feet was present.

A general and neurologic examination of the patient gave negative results. A roentgenogram of the right knee showed no abnormality. A diagnosis of gonorrheal arthritis of both knees and both feet was made. The tumor was considered to be either a lipoma or a fibroma, probably the former.

The tumor, which was removed with simple local anesthesia a few days later at the Hospital for Joint Diseases, arose from the region of the lower pole of the patella as a discrete mass of fat which slipped in and out of the joint, as noted on examination. There were marked hypertrophy and some inflammation of the synovia and fat near the fat pads. The synovia in general was definitely abnormal, in that it was red and hypertrophic in places. The joint cartilage was normal. Both fat pads were slightly enlarged. The external meniscus was normal. It was rather difficult to remove the tumor because it had not been transfixed. It was a true lipoma the origin of which was the external alar ligament.

The postoperative course was not eventful. Microscopic examination of the excised tumor proved its lipomatous nature. On May 29, nineteen days after the operation, the patient walked 7 miles (4.3 kilometers). On June 7, the right knee showed full motion and less crepitation than before. The tumor was no longer palpable. No complaints were referable to the knee.

Case 8.—The patient was a Negress, aged 39, who until seven months before her visit to the clinic was perfectly well. At this time, she fell while walking, striking her right knee. Pain was very mild at first, but this gradually increased in severity. The knee, which had become swollen at the time of the accident, remained swollen, and at the end of a month the knee was somewhat stiff. A month after this, the patient noted a lump in her knee, which moved about with motion of the knee. She had been able to climb stairs and to walk on uneven ground only by holding the knee stiff.

The right knee was about ¼ inch (0.6 cm.) larger in circumference than the left and showed a small effusion. A rounded mass was felt on the medial aspect of the patella, which slipped in and out of the space between the patella and the internal condyle of the femur as the knee was flexed and extended. The mass measured 1 by ¾ inch (2.5 by 2 cm.) and was smooth, rounded and definitely firm. In the course of its slipping it produced temporary locking, causing temporary severe pain. In the flexed position of both knees at an angle of 150 degrees mild lateral instability was present, with some bony grating.

General examination gave negative results except for a large uterine fibroid. Years ago she had received antisyphilitic treatment. Recently the Wassermann reaction of the blood had been negative.

A joint body, possibly a chondroma or fibroma, was thought of. It was also suggested, in view of the mild lateral instability with bony grating (in the flexed position), that a Charcot knee be considered. A roentgenogram of the knee did not show abnormalities, and this diagnosis was discarded.

On Jan. 24, 1934, an arthroscopic examination and arthrotomy were done. The arthroscopic procedure was done to determine the possibilities of visualization of such a tumor and also to determine the general state of the joint. Experience in the previous case had taught us to expect other intra-articular pathologic changes. Chronic villous synovitis was noted in the joint. The region of the fat pads and the bottom of the joint could not be well examined. The tumor on the medial aspect of the joint was visualized indistinctly, especially when it was forced into the joint on external pressure. Its edges could not be determined. Its surface was white with red streaks over it. The mass was much too large to be seen in one area of vision. In general arthroscopic examination of the joint was not very satisfactory.

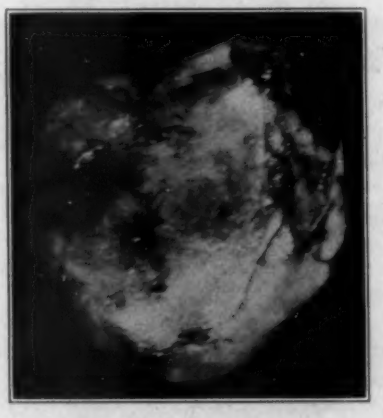

Fig. 13 (case 8).—A photograph of the lipoma removed from the knee joint.

The tumor was transfixed by two needles before the joint was entered and its removal was made easier by this transfixion. The tumor was encapsulated; It was the size of the yolk of an egg, and it entered the joint from the superior internal angle of the patella. Its exact point of origin was not determined through the small incision used for its removal. No note was made of changes in the synovia or of other pathologic change within the joint, and indeed the incision was much too small for a good view of the interior of the joint. The microscopic examination of the tumor showed an encapsulated growth of fatty tissue in the form of a slightly flattened disk 3.5 cm. in diameter. Histologically, it was a lipoma.

The postoperative course was uneventful. It was difficult to establish motion because of severe pain in the knee and because of persistent swelling. A roentgenogram made on March 22 showed marked atrophy of the bones of the knee joint. The angle of greatest extension, both actively and passively, was 180 degrees; flexion was almost normal then. No definite points of tenderness were

present about the knee. The patient felt the knee to be weak and therefore used crutches. On April 7 the angle of greatest extension was 180 degrees and the angle of greatest flexion was only 90 degrees. Attempt at further flexion was painful. In November 1934 the knee was no longer swollen, but it was still crepitant on motion. Flexion was almost full. The patient still complained of some pain. On her last visit, on April 20, 1935, she complained of pain in her knee on going up and down steps and in rainy weather. She walked with a slight right-sided limp. Motion was complete but slightly crepitant. The patient was satisfied with the end-result.

SYMPATHETIC SYNOVITIS OF THE KNEE JOINT ARISING FROM AN ADJACENT BONY FOCUS

CASE 9.—Le Roy D., a Negro aged 15, was seen in the clinic on Dec. 5, 1933, because of a swelling of his right knee, which had appeared recently without trauma. A year previously he had noted a similar swelling, which had promptly subsided. Examination of the joint after aspiration of 150 cc. of yellow synovial fluid gave essentially negative results. A Wassermann test of this fluid caused a reaction of 4 plus. On December 9, 60 cc. of synovial fluid was aspirated; on this occasion and on two other occasions shortly after that the Wassermann reaction of the fluid was negative. The Wassermann and Kahn reactions of the blood were also negative. The reactions to tuberculin were negative. This thorough serologic check-up caused us to discard a presumptive diagnosis of syphilitic arthritis. The patient was admitted to the Hospital for Joint Diseases on Jan. 15, 1934, because of disability of the knee. Slight pain was complained of, and some limitation of motion due to the accumulation of fluid was noted.

The patient walked with a mild right knee limp. The knee could not be extended fully, and the angle of greatest extension was 175 degrees, whereas the angle of flexion was 60 degrees. A patellar ballottement was present, and some periarticular thickening was noted. No tenderness was present in or about the joint. Definite pain was complained of over the tibia about 3 inches below the level of the joint line, especially about the posterior portion of the tibia. The muscle of the calf was tender. Heat and some swelling of the soft tissue were felt over the tibia, especially posteriorly. Tenderness was very marked about 4 inches (10 cm.) below the joint line, and a mass could be felt on the inner side of the tibia.

The latter findings had not originally been noted, nor were they present at the previous examination. A roentgenogram of the right knee, which had been made in the clinic, had been interpreted as showing no abnormalities, but on reconsideration at this time there was seen in the lowermost part of the tibia represented on the film, about 3 inches below the knee joint, a peculiar shadow of sclerosis. Other roentgenograms, which visualized the entire tibia, showed circular areas of density in the proximal one third of the bone projecting into the medullary cavity from the posterior portion of the substantia compacta. Sclerotic borders defined fairly well these areas of density. The cortex at the junction of the proximal and middle two thirds of the shaft was distinctly thickened. The periosteum was normal. The medullary cavity showed no further changes. No formation of new bone was noted in the soft parts.

A clinical diagnosis of abscess of the bone of the tibia was made, while a roentgenologic diagnosis of hemorrhagic osteomyelitis was given. The synovial effusions appeared to be due not to any intrinsic disease of the joint but to a sympa-

thetic irritation from the old but now active focus of disease in the bone. This opinion was held despite the distance of this focus from the joint, since no other diagnostic possibility presented itself.

Operation on January 17 by Dr. N. Ransohoff revealed a cystlike mass in the upper and outer posterior aspect of the tibia, lined with a raspberry-like membrane. Some fluid was encountered which looked like pus and which on smear showed a

Fig. 14 (case 9).—Note the cavitation in the posterior portion of the tibia. It is sharply demarcated by sclerotic bone. No evidence of adjacent periostitis is seen.

few pus cells but no organisms. An ostectomy was done. Culture of the tissues was sterile. A culture of one piece of bone was sterile, whereas a culture of a piece of another bone showed a micrococcus and a diphtheroid bacillus in forty-eight hours; these were probably contaminants.

Microscopically, spindle-shaped connective tissue cells were present as the predominant cell element, arranged compactly and often interlacing. Numerous drawn-out multinuclear cells were seen throughout sections of softer tissue. The discoloration of the tissue was due to blood pigment. A scattering of polymorphonuclear cells and mononuclear cells was seen. Some of the surrounding bone was sclerotic. The lesion in itself was not bone-forming. A pathologic diagnosis of an intramedullary fibrous tissue lesion, which might possibly be considered as a giant cell variant, was made.

The postoperative course was uneventful. Examination of the knee on February 1 demonstrated no patellar ballottement. On February 3 arthroscopic examination of the right knee was done to determine if any intra-articular lesion existed. Good visualization of the joint was obtained, and a moderate increase in fat was noted, especially in the form of large, irregular villi in the region of the fat pads. A thin, brown pannus was seen running vertically just above the intercondyloid fossa. No other significant findings were noted. The synovial change was considered to be the result of repeated effusion.

The limb was kept immobilized in a cast for three months, at which time the patient was allowed up. On May 5, after he had been up and about for a month, a return of effusion was noted in the knee. About 20 cc. was aspirated. The fluid was again aspirated, and on the last examination several months later fluid was still present in a small amount. The bony focus of disease was healing well.

As far as it can be determined in this case without actual arthrotomy, the effusion within the knee seems to have been due to the subjacent bony focus, a sympathetic synovitis. This condition is not uncommon and has occurred in many cases of juxta-articular lesions, including fracture, infection and tumor. Yet it was imperative here to rule out an actual lesion of the knee, and this was done by arthroscopic visualization.

SITE OF BENIGN GIANT CELL TUMOR ELEVEN YEARS AFTER OPERATION

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The literature contains few reports dealing with the sites of giant cell tumors long after operation, and there has been much curiosity as to the type of tissue filling the defects.

Herendeen [1] reported a series of six cases observed for from five to ten years in which the diagnosis of giant cell tumor was made from roentgenograms only. After roentgen therapy the symptoms disappeared, but subsequent roentgenograms revealed considerable abnormalities still existing in the bony structure. Although varying degrees of filling in by cancellous bone had occurred several years after the patients first came under observation roentgenograms still showed a picture which was atypical of giant cell tumor. No biopsies had been performed during the follow-up periods.

The purpose of this report is to record an instance of benign giant cell tumor of the upper end of the tibia in which histologic studies were made on material removed at the first operation and in which material for histologic study was obtained eleven years later at a second operation, performed on the same region because of recurrent pain and persistent abnormal roentgenographic findings of reduced density at the site of the tumor.

REPORT OF A CASE

W. H., a white man aged 42, was admitted to the service of Dr. D. B. Phemister at the Presbyterian Hospital on May 29, 1924. Roentgenograms of the right knee showed a well circumscribed oval area of reduced density involving the upper mesial aspect of the metaphyseal and epiphyseal regions of the tibia. Clinical diagnosis indicated a benign giant cell tumor. At operation the anteromesial aspect of the upper portion of the tibia was exposed through a linear incision, and the lesion was removed for the most part en masse, together with a portion of overlying cortex. The walls of the resulting cavity were then curetted and the wound closed without drainage. During the six months following operation ten roentgen treatments were given. A sinus discharging a serous fluid developed in the wound shortly after operation and persisted for several months, but it finally healed spontaneously.

From the Department of Surgery of the University of Chicago.

1. Herendeen, R. E.: Results in Röntgen-Ray Therapy of Giant-Cell Tumors of Bone, Ann. Surg. **23**:398 (Jan.) 1931.

Fig. 1.—Roentgenogram taken in May 1924 of a benign giant cell tumor in the upper portion of the right tibia.

Fig. 2.—Roentgenogram taken in March 1935 of the right knee joint, showing the appearance of the site of a benign giant cell tumor eleven years after operation.

The patient remained free from symptoms until the spring of 1934, when dull pain, related to physical activity, was experienced in the upper part of the right leg. It gradually became aggravated until March 1, 1935, when the patient was again seen. Physical examination revealed nothing abnormal except in the upper part of the right leg. Here the linear scar appeared healed; there was no erythema of the skin or visible swelling. On palpation there was some tenderness on the mesial side of the tibial tubercle, and the underlying bone felt roughened. The motions of the knee were not impaired. There was no evidence of excess fluid in the knee joint. Roentgenograms of the upper portion of the tibia revealed a rather large oval, multiloculated area of reduced density, with irregular longitudinal streaks of increased density and a dense cortex at the lateral margin of the lesion. When the new roentgenograms were compared with those taken eleven years previously, some reduction in the size of the lesion, due to irregular filling in by both dense and cancellous bone, was noted. The mesial cortex also appeared considerably thicker. The clinical diagnosis was either a recurrent benign giant cell tumor or recurrent osteomyelitis at the site of the tumor.

At the second operation, on March 2, 1935, the anterior surface of the upper 3 inches (7.5 cm.) of the tibia was exposed through a linear incision, and the periosteum was reflected mesially and laterally. A window 1½ by 1 inch (3.8 by 2.5 cm.) was excised through the cortex, exposing cancellous bone. There was no gross evidence of tumor. The cancellous bone was penetrated laterally, and an oval area filled with fibrous tissue was found and cleaned out. After more cancellous bone was removed mesially, a cavity about ½ inch (1.3 cm.) in diameter was entered and found to contain very spongy bone and fatty and fibrous marrow. The periosteum was then approximated by catgut sutures and the skin sutured with silk. The convalescence was uneventful.

Cultures of some of the tissue removed at operation remained sterile.

Histologic Examination.—Sections made in 1924 from the tissue then removed and more recent sections made from blocks cut from the preserved material revealed a typical benign giant cell tumor with rather large spindle cell stroma, throughout which were scattered numerous large foreign body giant cells. Some sections from the margins of the lesion, however, revealed nothing suggesting a benign giant cell tumor but showed instead a rather dense fibrous stroma in which were scattered numerous large fat-filled macrophages (foam cells). Such a picture is consistent with what has been described as xanthoma of bone. We have observed such areas in sections of marrow bordering various types of primary and secondary neoplasms in bone, and we feel that the appearance is due to degeneration of the fatty marrow as a result of direct pressure or of tumor infiltration or that it represents a late stage in the resorption of extravasated blood. Thus, sections from all portions of a lesion should be studied before the diagnosis of xanthoma of bone is made.

Sections of the tissue removed from the oval area on March 1, 1935, revealed a dense fibrotic marrow, in which fibroblast nuclei were not numerous. At the margins of this tissue there were coarse trabeculae of cancellous bone. The relation of the latter to the dense fibrotic marrow suggested that the areas of fibrosis were being gradually and very slowly replaced by cancellous bone such as filled the other cavity. The fibrotic areas also exhibited scattered small foci of small foreign body giant cells, with which were a few round cells. These cellular foci were about the border of the fibrous area and probably were associated with the chronic transformative change and did not represent remnants of the giant cell tumor. Such foci did not exhibit active cellular proliferation.

Sections of chips of cancellous bone showed normal fatty marrow and bony trabeculae.

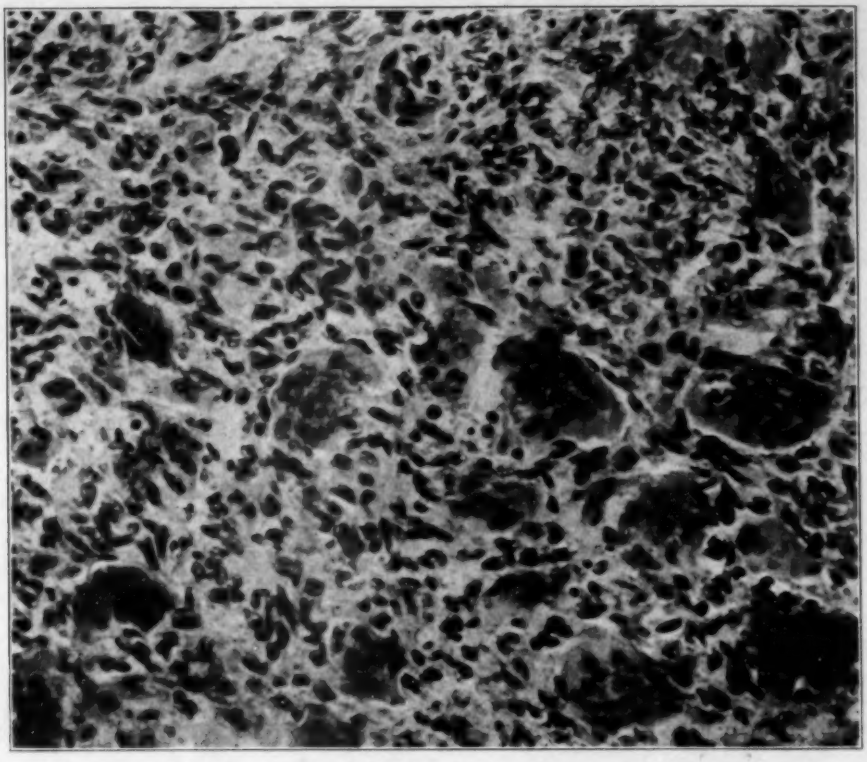

Fig. 3.—Photomicrograph of a section of a benign giant cell tumor removed in May 1934. Magnification, × 300.

Fig. 4.—Photomicrograph of a section of dense fibrous marrow removed from the site of a giant cell tumor in March 1935. A focus of small foreign body giant cells is seen. These are most likely a part of the chronic healing process, in which fibrous tissue is slowly being replaced by spongy bone and marrow. Magnification, × 300.

COMMENT

The case described illustrates that although a benign giant cell tumor is clinically controlled for many years, roentgenographic evidence of the lesion may persist. Ingrowth of cancellous bone at the site of the neoplasm may be a very slow process and may require many years for completion. In some instances complete restoration of the normal architecture of the bone may not be accomplished during the life of the patient. Persistence of reduced density in roentgenograms years after curettage does not therefore necessarily indicate persistence or recurrence of the neoplasm. If serial roentgenograms made over a period of months several years after the operation fail to show progression of the lesion and the patient remains free from symptoms, it may be assumed that the lesion is controlled. On the other hand, recurrence of symptoms, even though roentgenographic examination shows no progression of the lesion, justifies reexploration of the site of the tumor.

PARALYTIC DISLOCATION OF THE HIP

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The problem of therapy presented by paralytic dislocation of the hip causes every orthopedic surgeon many moments of uneasiness and doubt. Difficult as the treatment of this condition has always been, the dilemma in which the surgeons of an earlier generation found themselves could still be resolved more readily than that of the modern surgeon. In his review of the condition, Sever [1] divided cases of paralysis of the hip into three categories: The first group consisted of cases of the early stage, in which mild paralysis without dislocation existed. In these the prognosis was good, and the treatment consisted of the correction of deformities and the application of a plaster or a brace. The second group consisted of cases in which a reducible paralytic dislocation existed. The prognosis was not as good as that in the former group, but the treatment consisted of correction of the contractures and the application of a brace or a plaster. In the third group of cases, those of the irreducible paralytic dislocation, the prognosis was bad, and fusion was recommended after correction of the contracture. There was really no great problem, and no discriminating choice of procedure was either necessary or possible. But since the increase of the surgical horizon by means of the Jones operation, the Lorenz method of bifurcation, the Schanz osteotomy, the shelf operations described by Gill and Albee, the Dickson method of transplantation of muscle, etc., the choice of method has become serious and vital. The surgeon must constantly face the question whether conservative or radical treatment should be employed. In the event that operation is deemed necessary, he must determine whether mobility is invariably to be sacrificed in favor of stability or whether the effort to conserve both functions is justified.

From some of the reports in the literature the impression may be gained that the latter alternative is to be preferred to the former. Elzinga and Key [2] after a study of twenty-six cases, in eight of which an operation was performed, stated categorically: "The operation recommended is the shelf operation." Jones [3] reported four cases in which the opera-

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From the Hospital for Joint Diseases, service of Dr. Harry Finkelstein.

1. Sever, J. W.: Boston M. & S. J. **165**:313, 1911.
2. Elzinga, E., and Key, J. A.: J. Bone & Joint Surg. **14**:867, 1932.
3. Jones, Ellis: J. Orthop. Surg. **2**:183, 1920.

tion for the establishment of a bony shelf gave good results. On the other hand, Gill [4] in his paper on congenital dislocation of the hip reported nine cases of the paralytic type, in only five of which good results followed the shelf operation. Though other authors have reported occasional satisfactory results following one or another procedure, it is to be noted that the information on this subject is meager. The consequence of this actual dearth of knowledge is an uncomfortable feeling of uncertainty as to the method best indicated in any particular instance and is the justification for our brief review of the subject.

The series of twenty cases which we have been able to observe in the several orthopedic services of the Hospital for Joint Diseases is admittedly small. Yet the survey of the histories seems to point to certain conclusions which we believe to be of interest and of value.

Fig. 1 (case 1).—Roentgenograms showing bilateral flaccid paralysis of the hips with dislocation of the left hip. Note the tilt of the pelvis on the side of the dislocation and also the changes which occurred in the left hip after dislocation and the absence of such changes in the right hip despite the paralysis.

REPORT OF CASES

CASE 1.—A. B., a youth aged 16, was admitted to the hospital in July 1933, about three years after he was shot accidentally. As a result of this he suffered from transverse myelitis. Up to the time of the injury the patient had been well, but within two and one-half years a definite shortening of the left leg was observed. When he was admitted to the hospital bilateral paralysis of the hips was noted. The head of the right femur was in the acetabulum, and there was

4. Gill, A. B.: J. Bone & Joint Surg. **17**:48, 1935; personal communication to the authors.

a definite contracture of the adductor, the external rotator and the extensor muscles of the hip. The left hip was dislocated posteriorly, and there was a contracture of the flexor and adductor muscles. The pelvis was tilted upward on the left side. On the right side roentgenograms showed a good acetabulum, with slight changes in the head and neck of the femur; on the left side roentgenograms showed a poor acetabular shelf, with dislocation of the head of the femur posteriorly and changes both in the head and in the neck of the femur (fig. 1). A number of unsuccessful attempts were made at reduction of the dislocation by means of skeletal traction. The Roger Anderson type of traction was then applied to correct the pelvic obliquity, and tenotomy of the adductor muscles was performed. This was partly successful, and a Hessing brace was applied with the leg in abduction. The patient lived in another state and has not been heard from since.

Case 2.—M. S., a girl aged 10 years, was first seen in September 1933. At the age of 6 weeks she had whooping cough, with a cerebral complication which led to convulsions. At the age of 2½ years a cerebral blood clot was removed, and thereafter the patient made slight improvement. For the past several years the right hip and knee had been flexed and adducted over the opposite leg. About three weeks before she was admitted to the hospital the child suffered one of her periodic "attacks," during which the left leg went into spasm in extension, while the right leg was flexed. Shortly after this the mother noted that the right leg was becoming shorter. Examination disclosed a typical flexion-adduction deformity of the right leg, with dislocation of the hip. Roentgenograms showed a dislocation of the head of the right femur, which was deformed, and a shallow acetabulum with a poor acetabular roof; those of the left hip showed a slight flattening of the head, with a well formed acetabular shelf. An operation was not performed because of the general neurologic condition.

Case 3.—I. K., a boy aged 9 years, was admitted to the hospital in December 1933, with a history of spastic triplegia since birth. Tenotomy had been performed elsewhere several times, and the patient came under treatment primarily for the condition of the left hip. The left leg was held in marked adduction, while the right leg was in abduction and external rotation. Roentgenograms showed an apparently normal right hip, with dislocation of the left femur upward and forward. The acetabulum was shallow and irregular in contour, the acetabular shelf was deficient and the pelvis was tilted upward on the left side owing to weakness of the abdominal and spinal muscles. A shelf operation was performed, with transplantation of the tensor fasciae latae muscle, and thereafter turnbuckle casts were applied, with the object of maintaining the reduction. However, as soon as the traction straps were removed the deformity returned, in spite of the fact that an excellent shelf was to be seen. The roentgenogram made by Dr. Pomeranz showed that the acetabular shelf projected well beyond the head of the femur and extended down to the region of the great trochanter, encircling the proximal extremity of the femur. The head of the femur appeared to be subluxated slightly upward as compared with the plate made previously. Because of the low grade of mentality and the lack of cooperation, no further operative procedure was deemed advisable, and the patient was transferred to a convalescent home.

Case 4.—F. A., a boy, aged 8 years when he was first treated at the hospital in 1928, had been afflicted with poliomyelitis three years previously and had residual paralysis involving the back and both lower extremities. The spinal muscles on the right side were stronger than those on the left. There was a slight flexion deformity at the left hip but no evidence of dislocation. Except for

slight action of the extensor muscle, all the muscles of the right hip were powerless. The same was true of all but the external rotator muscles of the left hip. One ankle was stabilized, and plaster of paris bandages were applied to correct and maintain the correction in the spine and in the extremities. In November 1933 the patient returned to the hospital with a marked flexion contracture of the left hip. The pelvis on the left side was 3½ inches (9 cm.) higher than on the right side and was tilted markedly forward, so that the patient had severe scoliosis and lordosis. Roentgenograms of the right side showed a fairly normal head, neck and acetabulum, while on the left side the head was dislocated, the acetabular roof was poor and the femur was in varus. Because of the debility of the patient and the extent of the paralysis an extensive operation was not considered advisable. The pelvic obliquity was corrected by tenotomy and traction, and the patient was fitted with a brace and a crutch, with which he was able to get about.

Case 5.—H. F., a man aged 31, was admitted to the hospital in July 1933. He had poliomyelitis at the age of 1½ years. At first he was treated by means of a brace, but subsequently he was operated on and was able to get about very well until three years previously, when he slipped. Something "snapped" in his left hip, and since that time he noticed the progressive development of a limp. Examination disclosed a typical dislocation of the left hip posteriorly. The pelvis tended to sink on the left side. The gluteus medius muscle was not present, and the tensor fasciae latae muscle was weak, but otherwise the muscles about the hip were powerful. Roentgenograms showed a sloping acetabular roof and an elongated, widened acetabulum. The head of the femur was well formed but was lying posteriorly; the neck was in valgus and was slightly anteverted (fig. 2). An attempt was made to reduce the dislocation by skeletal traction, but the patient refused to tolerate the treatment and left the hospital against advice.

Case 6.—O. B., a boy aged 14, was admitted to the hospital on Nov. 2, 1929, with a history of infantile paralysis at the age of 6 months. Stabilizing operations were performed on his legs. When he was admitted to the hospital the patient walked fairly well. The left leg was 2 inches (5 cm.) shorter than the right leg. There was a flexion contracture of 30 degrees in the left hip. The adductor muscles were more powerful than the abductor muscles; the tensor fasciae latae muscle was good, and the hamstring muscles were somewhat more powerful than the quadriceps muscle. Roentgenograms showed a shallow acetabular cavity with a deficient shelf. The Dickson and Souter operations were performed for flexion contraction on November 20, and a plaster of paris cast was applied. In February 1931 the patient was admitted to the hospital for a bone-lengthening operation; roentgenograms showed the same type of marginal subluxation of the femur, but it was noted that the patient had good stability and so no further operation was performed on the hip.

Case 7.—G. J., a boy, was first admitted to the hospital in June 1931, at the age of 6½ years. Poliomyelitis at the age of 8 months left him with paralysis of the right upper extremity and both lower extremities. Examination disclosed a marked upward tilting of the pelvis on the right side, with severe lordosis due to a forward inclination of the pelvis. As was to be expected, the muscles of the spine and abdomen on the right side were much stronger than those on the left side. The function of the abductor muscles of the left hip was 2 plus; that of the adductor muscles of the right hip was 3 plus, and that of the right tensor fasciae latae muscle was 2 plus. The hamstring and flexor muscles of the hip were powerless. There was a flexion contracture of the right hip, which was dislocated. The left hip was flail. Without crutches the patient was unable to

stand. Roentgenograms showed a marginal subluxation of the right hip, with bony changes in the pelvis and femur. As a first step in treatment, the contracture of the abduction deformity of the left leg was undertaken, first by means of a plaster bandage and later by stripping the tensor fasciae latae muscle and the gluteus medius muscle from the ilium. Several months later the adduction-flexion deformity of the right hip was overcome by cutting the tensor fasciae latae and the rectus femoris muscles. A shelf was then turned down from the side of the ilium, and the tensor fasciae latae muscle was transplanted to a posterior attachment. Though this made a marked improvement in the patient's condition, the

Fig. 2 (case 5).—Roentgenogram showing unilateral paralysis, with a sloping acetabular shelf which was sufficient until trauma led to dislocation. Note the tilting of the pelvis downward on the affected side and the configuration of the head.

result was not considered satisfactory because of the persistence of some pelvic obliquity. This was ultimately relieved in 1934 by means of a plastic operation on the abdominal fascia. Roentgenograms showed the shelf in situ, with correction of the pelvic obliquity. Since his discharge from the hospital, the patient has been walking about with the aid of crutches in a satisfactory manner.

Case 8.—S. T., a girl aged 9 years, was admitted to the hospital in February 1930 with residual paralysis following poliomyelitis of six years' duration. Until one year previously, when she fell, the patient had been able to walk well with

the aid of a brace. Roentgenograms made at that time showed a dislocation of the left hip, and a plaster of paris bandage was applied. Examination in 1930 revealed a relatively mild form of paralysis. The flexor and extensor muscles of the hip, though reduced in power, were present and were of about equal power. The adductor muscles showed a slight predominance over the abductor muscles, but there appeared to be no definite pelvic tilt and no severe contracture. A typical shelf was turned down from the side of the ilium, and the tensor fasciae latae muscle was transplanted posteriorly in the manner described by Dickson. Roentgenograms showed a large, well formed shelf, with the head lying beneath it. The patient could not be examined recently but is reported to have a good result from the operation.

Case 9.—J. B., a girl aged 5 years, was admitted to the hospital in December 1927 with a history of poliomyelitis two and one-half years previously. The pelvis was inclined forward and definitely upward on the left side. All the muscles of the left hip except the flexor muscles and the tensor fasciae latae muscle were extremely weak. On the right, moderate power was noted in all the muscles except the extensor and adductor muscles. The right leg was held in flexion and abduction contracture, while the left was in flexion and adduction and was dislocated posteriorly. The tensor fasciae latae, the rectus femoris and the psoas muscles and the capsular ligament were contracted; so they were cut. The hip was reduced, and a spica was applied. Roentgenograms showed the reduced hip lying in a poorly formed acetabulum with an insufficient roof. On the right side the head and acetabulum showed only slight abnormalities. After her discharge, the hip was dislocated again, and in February 1928 the patient was admitted to the hospital for tenotomy of the adductor muscle. In 1930 marked lordosis and pelvic obliquity were noted, and the patient was admitted to the hospital for a shelf operation. Roentgenograms showed a well formed shelf with the head in the reduced position. The patient was discharged, walking with the aid of crutches and a brace.

Case 10.—F. B., a girl aged 14 years, was admitted to the hospital in January 1928, with a history of infantile paralysis twelve years before. Only the right leg was involved. The patient had had several previous operations on the ankle with manipulative reduction of the dislocation of the hip. The mother noted that the shortness of the right leg was increasing. When the patient was admitted to the hospital it was noted that there was a definite upward tilting of the pelvis on the right side. The function of the flexor muscles of the hip was 4 plus, that of the gluteus maximus muscle and the abductor muscles of the hip was 2 plus and that of the adductor muscles was 4 plus. There were about 2 inches (5 cm.) of shortening of the right leg, a definite flexion deformity at the right hip of 15 degrees and an adduction deformity of 30 degrees. Roentgenograms showed a dislocation of the right hip. In February 1928 a shelf operation was performed. Roentgenograms made in 1930 showed the pelvis to be somewhat oblique, but the hip was apparently beneath the shelf. In March 1930 the patient was able to walk with the aid of crutches and a brace, which were discarded shortly afterward.

Case 11.—E. R., a girl, was admitted to the hospital in May 1933, at the age of 12 years. At the age of 5 weeks she had been operated on for meningomyelocele and spina bifida. When 1 year old she was able to walk with the aid of crutches. Paralysis of the feet was noted as early as 1926, but the muscles of the hip were considered good. In 1929 it was noted that the child was walking well but that operations for the stabilization of both ankles were indicated. In 1930 it was noted that there was a dislocation of the left hip, and in 1931 both hips were

dislocated, the left leg being higher than the right. Good strength was noted in the adductor muscles. Roentgenograms showed bilateral dislocation of the hips, with no acetabular fossae. A shelf operation was performed on the left hip in May. Roentgenograms made one month later showed a fairly thick and large newly formed acetabular shelf, which extended well beyond the displaced capital epiphysis of the femur. Somewhat later it was noted that the acetabular shelf appeared to have slipped upward and slightly outward. The patient was discharged to a convalescent home in a plaster cast and at the time of writing was reported to be only slightly improved.

CASE 12.—L. B., a girl, was afflicted with infantile paralysis at the age of 1 year. She was left with residual bilateral paralysis of the extremities. Treatment before she was admitted to the Hospital for Joint Diseases consisted of the application of a double spica and later the stabilization of the left foot. With the aid

Fig. 3 (case 10).—Roentgenogram showing redislocation of the head despite two shelf operations.

of a brace on the left leg the patient was able to walk. When seen in 1929, at the age of 4, there was a flexion contracture of the left hip with practically no muscle power. The right leg was adducted and flexed. The tensor fasciae latae muscle was strong, and the abductor muscles were weak. Roentgenograms showed that in spite of a fairly good but somewhat sloping shelf there was an upward dislocation of the head of the femur on the right side. The adductor muscles were divided, the hip was reduced and a shelf was turned down superior to the head. The tensor fasciae latae muscle was transplanted posteriorly. The patient was discharged in a plaster cast and was subsequently fitted with a brace. In October 1930, somewhat over a year later, the patient was admitted to the hospital with a recurrence of the flexion of the left hip and dislocation of the right hip. The flexion of the left hip was corrected by means of the Soutter and Yount operations.

In December 1934 the right hip was operated on again, and an additional shelf was turned down posteriorly, but roentgenograms showed that in spite of an excellent roof marginal luxation of the head had recurred (fig. 3).

CASE 13.—A. L., a girl aged 17, was admitted to the hospital in February 1932, about fifteen years after an attack of poliomyelitis, which left her with residual paralysis involving both extremities. There were marked lordosis and scoliosis. The pelvis was inclined forward and was elevated on the left side. The muscles of the left hip showed fair power of flexion and adduction. All the other muscles were absent. The right hip was completely paralyzed, except that there was slight power in the abductor muscles and good power in the extensor muscles. Roentgenograms showed a well formed head articulating with a good acetabulum on the right side. It was deemed desirable to stabilize the foot before proceeding to the operation on the hip. Several months after this operation, the patient was admitted to the hospital for treatment of the condition of the hip. A shelf operation was performed on the left side in June 1932, though it was realized that the chances for a successful outcome were not good because of the absence of the abductor muscles. This proved to be the case. Four months after the operation the hip was completely dislocated and was unstable, and the shelf was found to have fractured. In December 1932 the patient was admitted to the hospital for a bifurcation operation. Roentgenograms showed firm union at the site of the osteotomy, with reunion of the fragments of the fractured acetabular shelf. When the patient was examined about six months later she was walking with the aid of crutches. The hip was stable in 15 degrees of abduction, and the patient was satisfied with the strength of her leg.

CASE 14.—F. K., a girl, was first treated in 1926, at the age of 8 years, for severe bilateral paralytic clubfoot and paralytic dislocation of the hip due to spina bifida. The pelvis was tilted upward on the left, and the left leg was consequently in adduction, while the right leg was in slight abduction. There was practically no power in either extremity. Roentgenograms showed dislocation of the left femur, with poor formation of the shelf. The head of the right femur was in the acetabulum, with marked deformity of the neck and a change in the direction of the capital epiphysis. Operations were performed for stabilization of the ankles, and in January 1927 a new acetabulum was reamed out. Though the head seemed to remain in place in all positions of the leg when tested on the operating table, roentgenograms made several months later showed a recurrence of the dislocation and an increase in the pelvic obliquity. To correct this, the abductor muscles of the right hip and the adductor muscles of the left hip were tenotomized. Thereafter the left hip was fused, and the patient was discharged, walking with the aid of braces and crutches. The patient died about six years later of an intercurrent infection.

CASE 15.—F. O., a boy aged 7 years, was first admitted to the hospital in June 1934, five years after an attack of poliomyelitis. Only the right leg was involved. The pelvis was tilted downward on the affected side, and there was a moderate increase in its forward inclination. The abdominal and the spinal musculature were normal. The abductor muscles of the right hip were completely paralyzed, and the hip was in definite flexion and adduction contracture. Roentgenograms showed a poorly developed acetabulum, with an inadequate acetabular roof. A shelf was turned down, and the tensor fasciae latae muscle was transplanted. Roentgenograms made about six months later showed a recurrence of the upward luxation of the femur. On the patient's admission to the hospital in May 1935 the flexion contracture had become reestablished, owing to the weakness of the gluteal

and abductor muscles and the tensor fasciae latae muscle and to the unopposed action of the sartorius muscle and the adductor muscles. Fusion of the hip was performed, and the patient is still under observation.

Case 16.—N. C., a girl, was admitted to the hospital in 1926, at the age of 12 years, suffering from the effects of residual poliomyelitis acquired at the age of 2 years. She had submitted to several operations for stabilization of the ankles. Since our primary interest lies in the hip, the record of the other treatment will be omitted. When the child was 5 years old the mother noted the development of a progressive limp on the right side. When she was first seen, the right hip was flexed and adducted and showed a typical iliac dislocation. The hip was reduced by traction, and a spica was applied and later a brace. The patient was given abduction exercises, but in 1927 roentgenograms showed redislocation, and a shelf operation was performed. In 1929 the patient again showed symptoms, and roentgenograms showed marginal subluxation of the head. In 1934 the patient again returned, complaining of a limp. At this time marked improvement of the abductor muscles was noted, but the head was markedly displaced, and fusion was decided on. The head was pulled down a distance of about 1 inch (2.5 cm.) and was fused to the ilium in marked internal rotation. Later, to correct this, supracondylar osteotomy was performed. When seen in the outpatient department about six months later, the patient was walking without the aid of a crutch or brace. There was excellent stability, with a barely perceptible limp. Roentgenograms showed firm bony ankylosis at the hip, with union at the site of the supracondylar osteotomy.

Case 17.—M. K., a woman aged 21, was admitted to the hospital in June 1933. As a consequence of poliomyelitis at the age of 2, the patient had residual paralysis of the left leg and dislocation of the left hip. All the muscles of the hip except the sartorius muscle were absent. The left extremity was about 1 inch (2.5 cm.) shorter than the right, and there was a definite tilt of the pelvis downward on the left. Roentgenograms showed a dislocation of the hip, with marked underdevelopment of the osseous structures. A combined intra-articular and extra-articular fusion was performed, and the patient left the hospital wearing a plaster bandage. Subsequently the patient suffered a fracture through the fused hip. This was treated by means of Kirschner wires and healed with the leg in slight adduction. Roentgenograms showed bony union at the site of fracture and fusion of the hip. The patient submitted to operation for stabilization of the foot and in June 1934 was discharged walking and in good condition.

Case 18.—P. W., a girl, was first treated in 1927, at the age of 8 years, for complete flaccid paralysis of the right upper extremity and both lower extremities and marked paralytic scoliosis following infantile paralysis six years previously. At this time there was no dislocation, and plaster casts and later braces were applied. In 1929 an upward tilting of the pelvis on the left side was observed, and traction was used in an effort to combat the pelvic obliquity. In 1931 the patient was admitted to the hospital for treatment of dislocation of the left hip. The abdominal and the spinal muscles were very weak. The right hip showed a faint trace of muscle power; on the left side the function of the adductor muscles was 2 plus, and the flexor muscles showed a trace of power. The left hip was adducted and the right abducted. Roentgenograms showed dislocation of the left hip. An operation for fusion was performed in December 1931, but the result was not satisfactory. Instead of fusion, there was a wide shelf, which, however, did not function to restore stability of the hip. In November 1932 the hip was refused, and

the patient was discharged to a convalescent home. It was reported that, considering the extent of the paralytic involvement, the patient seems to have shown improvement after the second operation for fusion.

CASE 19.—S. L., a girl aged 14 years, was admitted to the hospital in October 1933, ten years after an attack of infantile paralysis. The residual paralysis was mild. The function of the flexor muscles of the left hip was 4 plus, that of the adductor muscles was 4 plus and that of the abductor muscles was 2 plus; function of the extensor muscles was nil. The muscles of the back and abdomen were strong. The leg was held in flexion and adduction, but the pelvis was tilted downward on the left side, the side of the dislocation. This, we believe, is accounted for by the shortening of about 1½ inch (3.8 cm.) which existed in the left lower extremity. In January 1935 fusion of the left hip was performed, but the graft did not "take." The further outcome cannot be reported at present, since the patient is to be operated on again.

CASE 20.—S. B., a woman, was admitted to the hospital in 1932, at the age of 23. She had apparently been treated for a congenital dislocation of the right hip at the age of 6 years. At the age of 11 years the patient had poliomyelitis, which left residual paralysis in the right hip. On examination the pelvis was found to be tilted upward on the right side, and there was apparently a flexion and adduction contracture. An attempt at fusion of the hip was made without success. Roentgenograms showed a shallow acetabulum, with a poor roof. The head was dislocated, and at the upper end of the shaft there was a peculiar hooklike process, evidently the remnant of the bone graft. The patient is to be admitted to the hospital again for operative fusion.

COMMENT

When we analyzed these cases, a number of interesting observations were made. In an active hospital service, a record of only twenty cases could be found over a period of ten years. In the twenty cases there were twenty-one luxations or subluxations of the hip. Though in ten of the cases there was bilateral paralysis of the hip of varying degrees of severity, bilateral dislocation was noted in only one (that of a patient suffering from severe spina bifida). In many cases the tensor fasciae latae muscle possessed considerable power, sufficient so that at operation the muscle warranted transplantation. In its anatomic position this muscle seemed to function more as a flexor than as an abductor muscle of the hip. In practically all cases in which dislocation occurred, the abductor muscles were either absent or so weak as to be valueless as stabilizers of the hip.

In sixteen of the twenty cases, various types of operation were performed. In seven cases the shelf operation alone was performed. Of these, a good result occurred in only two. Of the others, fusion was performed later in two, with good results; an operation for bifurcation gave a good result in one, and in another establishment of an additional shelf and a Dickson operation were employed, with an unsatisfactory outcome. Of the four cases in which the combined shelf and Dickson operations were performed, in two there was a poor result

(in one case, as mentioned previously the procedure was done on reoperation) and in two the result was satisfactory after a secondary procedure designed to correct the pelvic obliquity. In the one case in which primary deepening of the acetabulum was attempted the operation was unsuccessful, and an operation for fusion was subsequently performed, with a good result. In seven cases fusion was undertaken. In four of the cases fusion was a primary procedure. The result in one was good, in one refusion was necessary and in two the operation was unsuccessful. In three cases fusion gave a good result after the failure of other surgical measures.

In by far the great majority of cases reported the origin of the condition was poliomyelitic. However, this is not invariably true. Nolot [5] called attention to the fact that paralytic dislocations have been noted in persons with locomotor ataxia and with Pott's disease, with injury to the underlying cord. In our series, in cases 5, 9 and 12 the condition was due to spina bifida; in case 1 it developed following a bullet injury to the cord, with subsequent transverse myelitis; in case 2 there was spasticity in a patient who was obviously suffering from some cerebral disorder, probably hemiplegia following pertussis, and in case 3 the condition was due to a cerebral injury at birth. The latter cases are of unusual interest because dislocation of the hip occurs rarely, if at all, in any of the large group of patients with hemiplegia afflicted with paralysis of cerebral origin. In fact, while dislocations are seldom observed in persons with paralysis which results from lesions of the upper motor neurons, they seem to be typical in persons in whom the lower motor neuron arc is interrupted. The explanation of this remarkable preponderance is undoubtedly to be found in an appreciation of the elements which determine the stability of the hip joint.

Normally this depends on two factors; the character or competence of the acetabular block and the integrity of the pelvitrochanteric musculature. To insure perfect stability, both of these forces must be cooperating, and the loss of either predisposes to instability and dislocation. Of the two, the muscular element is, in our opinion, the more important. This does not in any manner minimize the importance of the acetabular shelf. From the experience gained by surgeons in the treatment of congenital dislocation of the hip, the significance of the shelf has been well attested. It is evident, however, that the data so obtained merely indicate the function of the shelf in the presence of otherwise good muscle power. In cases of paralysis the situation is markedly different, and the application of conclusions reached from the treatment of congenital dislocation to the problem of paralytic

5. Nolot, T.: Contribution à l'étude de la luxation paralytique de la hanche, Thèse de Paris, no. 326, 1905, p. 7.

dislocation is fraught with danger unless reservations are made. Thus, in our cases 3, 7, 11, 12, 13, 15 and 18 dislocation occurred in spite of excellent formation of the shelf. On the other hand, the patient in case 5, in whom a sloping shelf was noted, had no difficulty with his hip until the occurrence of a fall. This can be interpreted only as indicating that the stabilizing forces were sufficiently powerful, in spite of the paralysis and the sloping shelf, to prevent dislocation until such time as the additional trauma balanced the scales in favor of the dislocating forces. Elzinga and Key also noted that the size of the shelf or even its character does not seem to determine the stability of the hip. They stated: "We believe that an accurate placing of the shelf is more important than its size, as we have found small shelves just as efficient as large ones." Just where the shelf should be placed to be accurate was not stated, and in our opinion the "accurate placing of the shelf" can acquire significance only if by this term is meant the placing of the shelf so that it tends to fortify the stabilizing forces or to oppose the dislocating forces.

Unfortunately, Elzinga and Key did not state in particular the condition of the muscles in those cases in which good surgical results were obtained. They noted, however: "In eleven of twenty-six cases dislocation occurred in the presence of definite power in the extensor group. Much the same is true of the external rotators. Apparently the muscles which are most important in lending stability to the hip are the abductors and the tensor fascia femoris, as dislocation occurred in only two cases with fair abductors, in only three cases with a fair tensor fascia femoris and in no case in which these muscles were good or normal." This brings us to a consideration of the second element, the muscle factor, in the stabilization of the hip joint.

Apart from their function in moving the thigh, the pelvitrochanteric muscles act to maintain the contact of the head of the femur against the fulcrum of the acetabulum. When this contact is not maintained, as in the cases in which there is marked effusion into the joint or as in the cases cited here, the dislocation may occur regardless of the normal configuration of the bones. On the other hand, when this contact is maintained, as in cases in which spasticity of the muscles are associated with paralysis of the type caused by involvement of the upper motor neurons dislocation is among the greatest of rarities, though it does occur when the resultant forces are of sufficient magnitude. With paralysis caused by involvement of the lower motor neuron in which the muscles are completely atonic and contact cannot be maintained, dislocation will occur (as has been indicated) regardless of the nature of the shelf, while in the less severe cases, in which some muscle power still exists, especially in the extensor or abductor muscles even an apparently

insignificant shelf may be adequate to prevent dislocation. The important factor is the condition of the muscles about the hip. It is our belief that the dislocation of the hip will or will not occur in obedience to the resultant of forces acting on the hip and that the bony changes observed in the head and acetabulum of a dislocated hip are the result and not the cause of the dislocation, as Murk Jansen [6] demonstrated in cases of congenital dislocation.

Karewski,[7] Tillaux [8] and others [9] have demonstrated that when the resultant of forces is such as to lead to flexion, abduction and external rotation of the thigh, there is a tendency toward pubic dislocation. When the thigh assumes the position of flexion and adduction, the tendency is toward iliac dislocation. This is valid, whether the dislocation is of the congenital, traumatic, postinfectious or paralytic variety. The paralytic dislocation is merely a special condition due to the loss of power in some or all of the pelvifemoral muscles. This theory of the mechanism of dislocation by the unopposed action of certain groups of muscles was originally propounded by Réclus [10] and has been generally accepted by those who have given the matter thought. On the other hand, as Riedinger [11] and others have observed, accurate knowledge of the extent of the paralysis is in itself insufficient to warrant prediction of the exact type of dislocation which may be expected in any instance. This is due to the varying association of other factors, such as habitual posture, gravity, weight bearing and trauma. This was pointed out by Bradford,[12] Elzinga and Key [2] and others and was well demonstrated in our case 4. Our patient, though almost completely paralyzed, had a flexion contracture and a dislocation as a result of his position in bed. The condition in case 5, on the other hand, illustrated very well the effects of even minimal trauma in precipitating dislocation in a hip the stabilizing elements of which had been largely reduced by the effects of antecedent poliomyelitis. All of these forces are such as to increase the attitude of flexion and adduction and consequently predispose to dislocation.

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6. Jansen, Murk: J. Bone & Joint Surg. **11**:461, 1929.

7. Karewski, F.: Ann. Surg. **10**:226, 1889.

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9. Cramer, K.: Arch. f. Orthop. **9**:26, 1910. A good bibliography is included. Hoyt, W. A.: Ohio State M. J. **25**:962, 1929. Silfverskiöld, Nils: Hemiplegia Spastica Infantilis, Stockholm, P. A. Norstedt & Söner, 1924. Winterstein: Deutsche Ztschr. f. Chir. **242**:357, 1933.

10. Réclus, P.: Rev. mens. de méd. et de chir. **2**:176, 1878; Gaz. hébd. de méd. **20**:781, 1883.

11. Riedinger, J.: Arch. f. Orthop. **8**:270, 1910.

12. Bradford, E. H.: Boston M. & S. J. **108**:73, 1883.

When we speak of flexion and adduction being the dangerous attitude, we mean commonly flexion and adduction of the thigh in relation to the pelvis. But unless we speak of flexion and adduction in respect to a level pelvis, the use of the term is meaningless. It then becomes apparent that if we can draw a conclusion from the attitude of the flexion and adduction of the thigh in relation to the pelvis, we must conclude also that the same ill effects will result from the attitude of forward inclination and upward tilting of the pelvis in relation to the femur. This follows from the fact that flexion of the femur is the equivalent of forward inclination of the pelvis, while upward tilting of the pelvis is the equivalent of adduction of the femur. That these effects may be caused by different groups of muscles complicates the picture but does not alter the fundamental validity of the relationship.

In an excellent paper on the subject of pelvic obliquity, Mayer [13] called attention to the fact that the effective forces in causing a tilting of the pelvis in the lateral plane are: (1) contracture of the abdominal and spinal muscles, (2) contracture of the abductor muscles and (3) contracture of the adductor muscles. These three forces, either alone or in effective combination, determine a moment of rotation of the pelvis which results in the five types of lateral pelvic tilting which Mayer described. In a similar manner, tilting of the pelvis in an anteroposterior plane or inclination of the pelvis is controlled by the opposing action of the abdominal and spinal muscles and the flexor and extensor muscles of the thigh. Thus, forward inclination of the pelvis or lordosis may be caused by (1) contracture of the spinal muscles or (2) contracture of the flexor muscles of the thigh and the extensor muscles of the knee.

Except in cases 5, 15, 17 and 19, our patients exhibited a definite upward tilting of the pelvis on the side of the dislocation. In cases 17 and 19 this variation was readily accounted for by the actual shortening of the limb. This tendency to upward tilting is remarkable in view of the fact that in other forms of dislocation the tilting is usually downward on the affected side because of the relative shortening of the limb. Almost all showed, in addition, a marked degree of forward inclination of the pelvis. That this may have been partly due to the posterior dislocation of the fulcrum is immaterial, since the effect of this is merely to increase the efficiency of the forces causing the forward inclination. It leaves unaltered the general conclusion that the pelvic tilting in the anteroposterior and in the lateral plane is due to imbalance of the controlling musculature. Unfortunately, this condition does not seem to have aroused the interest which its importance would seem to merit. In cases 1, 4, 7 and 14, in which special attention was directed

13. Mayer, L.: J. Bone & Joint Surg. **13**:1, 1931.

to its correction, the results seemed to warrant the additional effort. It is possible that neglect in its treatment may account in part for the failures which have been recorded in the treatment of paralytic dislocations.

On viewing the problem of the therapy of paralytic dislocations in the light of the considerations previously noted, it will be seen to be much more complicated than was at first sight apparent. While a mild type offers no insurmountable difficulties, a complicated type may necessitate the solution of several auxiliary problems, each of which may be of major magnitude. The first of these is the question of deformity. To be sure, deformity should be prevented by appropriate application of plaster or a brace, but, willy-nilly, deformities do present themselves and they must be corrected. Not only must the deformity of the hip be corrected, but, of much more importance, the pelvis must be leveled. This may involve complicated surgical maneuvers: traction, application of plaster or a brace, various forms of myotomy or tenotomy, plastic operation on the fascia for paralysis of the abdominal muscles or even spinal fusion. The results may be questionable, yet the neglect of this factor may vitiate adequate treatment of the condition.

Once the pelvis is level, the question of treatment of the condition in the hip itself may be broached. This treatment may be conservative or radical. The conservative measures embrace the use of plaster or of abduction braces. A radical measure may consist of simple myotomy or tenotomy or may involve the performance of a complicated operation. The radical measures may be classified as operations on:

1. The capsule of the hip joint
   (*a*) Reefing or
   (*b*) Overlapping, as in the Jones operation
2. The bones of the hip joint
   (*a*) The acetabulum: a shelf operation
   (*b*) The neck of the femur: Lorenz, Hass or Schanz osteotomy
3. The joint
   (*a*) Arthrodesis
4. The muscles
   (*a*) The Dickson operation
   (*b*) Transplantation of the gluteal muscles
   (*c*) Tenodesis (e. g., using the tensor fasciae latae muscle)

In the matter of treatment of the first and third categories, established by Sever, most observers are agreed. In the case of mild involvement without dislocation, treatment should be such as to prevent dislocation. This may be done by elevation of the shoe on the opposite foot, by the application of plaster or a brace or by the performance of myotomy on the affected leg. All these measures are designed to

maintain abduction and extension and so to combat the development of the pernicious attitude of flexion and adduction. In the case of severe involvement treatment should be by arthrodesis. We are convinced, with Gill, that in such cases "arthrodesis is the operation of choice" and that when there is complete paralysis of all the muscles of the hip the shelf operation cannot be expected to give a good result. However, not every attempt at arthrodesis is followed by ankylosis of the hip joint; in adults even more than in children this tendency toward failure is pronounced. With this in mind, Sir Robert Jones [14] suggested a procedure which he considered successful. This consisted in relief of the contracture by means of fasciotomy, reduction of the dislocation, plastic fixation of the posterior portion of the capsule and then application of a plaster bandage, with the leg in abduction and extension, over a period of six months. We have had no experience with this method.

It is in the treatment of the condition in Sever's second category, in the cases in which there is reducible dislocation with some residual power, that the greatest divergence of opinion arises. Many authors consider the shelf operation the method of choice, but our own experience does not seem to warrant this feeling of assurance. Though the operations in our series were performed by various surgeons, as to whose skill no question can be raised, the results obtained by the shelf operation, even when combined with the stabilizing effects of the Dickson operation, were far from satisfactory. It is palpably impossible to reconcile such results with those reported by Elzinga and Key except on the basis of a difference in the choice of cases. From the experience gained in case 11, in which the shelf operation was originally opposed on the ground of absence of the gluteal muscles and in which subsequent operation for bifurcation gave a satisfactory result, we may perhaps be permitted to join in Gill's opinion that the shelf operation should be used only when there is evidence of fair power in the gluteal muscles. In other cases the various procedures used in the treatment of congenital dislocation may be attempted with the knowledge that in the event of failure arthrodesis is still held in reserve.

222 West Seventy-Seventh Street.

123 West Eighty-Sixth Street.

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TREATMENT OF GASTROJEJUNOCOLIC FISTULA BY MULTIPLE STAGE OPERATIONS

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HISTORICAL REVIEW

Gastrojejunocolic fistula is one of the rare complications attending gastric surgical procedure. Czerny [1] reported the first case in 1903. Bolton and Trotter in 1920 added four cases to the thirty-four already reported in the literature, while Judd [2] found records of only six cases at the Mayo Clinic previous to 1921. Rankin, Bargen and Buie [3] in their book published in 1932 attested to the rarity of the lesion. Lahey [4] in 1933 reported the observation of only six cases.

Since 1881, when Wolfer introduced the operation of gastroenterostomy, surgeons have had complications following gastric operations. Of the late complications following gastroenterostomy, none is more disappointing or more feared by the conscientious surgeon than the development of jejunal ulcer. Walton [5] stated: "As soon as it is evident that a gastrojejunal ulcer is present, an operation must be performed, for the condition is certain to progress and will give rise to pain, dangerous hemorrhage, or perforation." If the ulcer perforates into the peritoneal cavity, death from spreading peritonitis usually ensues; if the ulcer perforates into the transverse colon distressing diarrhea usually develops, with nausea, fecal vomiting and rapid losses of weight and strength from inability of the patient to obtain adequate nourishment. In Paterson's [6] cases the mortality in those in which operations were not performed was 100 per cent.

My paper briefly reviews the etiologic factors and the physiologic changes accompanying the lesion and describes the generally accepted methods of treatment. A case is reported in detail in which the procedure was successfully carried out in multiple stages, with the Mikulicz procedure.

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ETIOLOGY

Many theories have been advanced as to the probable cause of a simple gastrojejunal ulcer. The earlier operators felt that undue trauma to the mucous membrane of the jejunum and faulty technic caused the lesion. Nonabsorbable sutures and clamps were first held responsible for the formation of the new ulcer. Repeated attempts to cure the condition by local excision of the jejunal ulcer resulted in recurrence in over half the cases. The substitution of absorbable catgut ligatures for silk sutures failed to prevent the condition. Equally unsatisfactory results followed various forms of gastro-enterostomy. The patients showed marked hyperacidity and a tendency to the formation of ulcers.

TREATMENT

Until recently the only operation which seemed to give satisfactory results has been partial resection of the stomach, in an attempt to remove a part of the acid-bearing glands. Walton wrote that this operation is one of the most difficult and severe in modern surgery, for not only has the old anastomosis to be freed and separated but the gastrectomy should be extensive, and the ulcerated portion of the jejunum must be removed. The resultant mortality has been very high, ranging from 10 to 40 per cent, with recurrence in over half the surviving patients. With recent studies showing that the gastric glands near the pylorus produce an alkaline rather than an acid secretion, it is evident that a large part of the central portion of the stomach must be removed to lower the acidity to an appreciable degree. During the past decade gastro-enterologists have made distinct advances with diet and chemotherapy. Since they can now accurately control the levels of gastric acidity, it is seldom that an extensive gastric resection, with its attendant dangers, is warranted solely to control acidity.

When the gastrojejunal ulcer is complicated by a fistulous tract leading to the colon, the surgical work is increased, and the dangers of peritonitis from fecal contamination are very great. It is no wonder that such an experienced operator as Lahey wrote: "I approach gastrojejunal ulcer with colonic fistula still with hesitation and fear as to the question of possible fatality."

PHYSIOLOGY

Harvey Cushing[7] demonstrated many years ago that the gastric and duodenal contents were relatively sterile in normal persons and that bacteria increased in numbers and virulence as one progressed

7. Cushing, Harvey, in Howell, W. H.: Textbook of Physiology, ed. 11, Philadelphia, W. B. Saunders Company, 1930.

down the intestinal tract. Horsley [8] concluded that pathogenic bacteria do not grow in the normal stomach because of the presence of hydrochloric acid and that because hydrochloric acid is not present in the gastric juice of patients with carcinoma of the stomach bacteria do grow. For ten years Horsley fed his patients with gastric carcinoma or any other gastric disturbance associated with low acidity dilute hydrochloric acid for several days prior to operation to render the gastric content sterile. It is well known that peritonitis from the spill of normal gastric or duodenal contents is rare, while the slightest contamination of the abdominal cavity by the contents of the large intestines results in peritonitis, usually fatal to debilitated patients.

DISTURBED PHYSIOLOGY

Perforation of a gastrojejunal ulcer into the transverse colon produces certain physiologic changes. If the opening into the large intestine is small, very little food passes through it, most of the food taken by mouth being allowed to pass naturally through the entire length of the small intestine. Ample opportunity is thus provided for the absorption of nourishment. There may be only occasional symptoms referable to the gastro-intestinal tract for some time. If there is considerable inflammation or active ulceration about the opening, there may be intermittent periods of bleeding, nausea and vomiting, accompanied by pain and diarrhea. If the opening into the colon is large, much of the food passes directly from the stomach and jejunum into the transverse colon and does not have an opportunity to come in contact with the small intestine. Large quantities of nourishing food may be eaten but are of no value in supplying nutrition, as there is no opportunity for digestion and absorption by the small intestine. Loss of up to 75 pounds (34 Kg.) in weight has occurred from the inability of the patient to absorb the food ingested. Anemia and extreme weakness rapidly develop.

Another disadvantage of the direct opening from the stomach into the colon is the appearance of virulent bacteria in the stomach and upper part of the intestinal tract. A normally sterile field is changed into a potentially septic one by the appearance of colon bacilli in the stomach and jejunum. These artificial openings, which permit unusual bacteria and alkaline digestive juices to enter the normally acid stomach, frequently cause severe gastritis. On the other hand, the sudden appearance of undigested food in the transverse colon often results in the most troublesome diarrhea.

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8. Horsley, J. S., in Lewis, Dean: Practice of Surgery, Hagerstown, Md., W. F. Prior Company, 1929, vol. 6, chaps. 3 and 12.

SURGICAL RISK

Gastrojejunocolic fistula combines the hazards present in gastric operation with all the risks involved in surgical procedures on the colon. Usually the patient is dehydrated, emaciated and anemic. Surgical shock, hemorrhage, peritonitis and infections of the respiratory tract are the chief enemies. Every possible effort should be made to reduce these hazards. Prolonged operations, hours of general anesthesia and excessive handling of the viscera must be avoided. Scrupulous and exacting attention must be given to every detail of technic. The intravenous administration of dextrose, transfusions of blood and the ingestion of large quantities of carbohydrate in the form of fruit juices will frequently change the dehydrated, undernourished, anemic patient into a fair surgical risk.

PERITONEAL IMMUNIZATION

A preliminary injection of a concentrate of amniotic fluid, advocated by Young,[9] is a simple, rapid, nonhazardous and effective method of increasing peritoneal resistance. This procedure promises to supplant the more troublesome, painful and time-consuming method of introducing colon bacilli and streptococci as an intraperitoneal vaccine, with which Rankin and Bargen so ably pioneered. I have injected amniotic fluid many times without the slightest discomfort to the patient. Rarely, the fluid spreading throughout the general peritoneal cavity has caused slight nausea, but many times the patient has not been fully aroused from sleep by the procedure. Within a few hours there is usually a rise in temperature of 1 or 2 F., accompanied by a leukocyte count from 15,000 to 20,000 and an increase in the leukocytic content of the peritoneal exudate, which becomes turbid. This increase in the activity of the peritoneal exudate is held by Young [10] and Johnston to be direct evidence of the effectiveness of the immunization.

NEW APPLICATION OF THE MIKULICZ PROCEDURE

Recently I have used the following method of dealing with gastrojejunocolic fistula with satisfactory results. The method is reported in the hope that it may be of help to others faced with a similar problem. While doubtless other surgeons have employed multiple stage operations for this distressing condition, no report has been found in the literature of utilization of the Mikulicz procedure in this type of case. Extraperitoneal resection of the large intestine has increased the margin of safety in surgical intervention in cases of malignant disease of the colon. Application of the same principle should lower the mortality in cases of gastrojejunocolic fistula.

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9. Young, E. L., and Marks, G.: Surg., Gynec. & Obst. **59**:610 (Oct.) 1934.
10. Young, E. L.: Personal communication to the author.

REPORT OF CASE

History.—A 58 year old rancher was admitted to the hospital complaining of severe diarrhea of seven weeks' duration, with marked loss of weight and strength. Fifteen years previously a gastro-enterostomy had been performed elsewhere for a duodenal ulcer. Following this operation the patient was free from symptoms for eight years. For the past seven years the patient had noted some gas and burning epigastric distress after eating. This had been partially relieved by baking soda but had never been so severe as to prevent work. Three years previously the patient first noted attacks of diarrhea, occurring chiefly at night. At first these attacks were of from three to four days' duration and occurred at monthly intervals. The attacks gradually increased in frequency and severity. For the preceding two years the patient had been unable to do any work. Seven weeks before he was admitted to the hospital the diarrhea became constant, with from eight to ten stools daily, composed chiefly of mucus and undigested food. Blood had never been noted in the stools, but a marked burning sensation in the rectum had been present. The patient had lost 30 pounds in the past seven weeks and had been confined to the house because of weakness.

The patient's history, except for his gastro-intestinal complaints, was uneventful. He had always been a heavy smoker, but otherwise his habits were not remarkable.

Physical Examination.—Physical examination revealed the patient to be well developed but poorly nourished, somewhat dehydrated, with evidence of recent loss of weight. Aside from a large right inguinal hernia and the healed scar of the previous laparotomy, the physical findings were normal. The blood pressure was 130 systolic and 80 diastolic.

Laboratory examination revealed normal urine. The stools did not show blood and parasites but did show large quantities of undigested food. The blood picture was that of mild secondary anemia. Gastric analysis revealed marked hyperacidity: free hydrochloric acid 70 and total acidity 80.

Roentgen Examination.—Fluoroscopic examination by Dr. M. J. Geyman at the Cottage Hospital revealed a somewhat highly placed gastro-enterostomy stoma that appeared to be irregular. Adjacent to the opening a fleck of barium sulfate appeared, suggesting an ulcerated margin. The duodenal bulb was deformed and contracted, but there was no evidence of an active ulcer of the duodenum. Most of the meal appeared to leave the stomach by the gastro-enterostomy opening. In less than thirty minutes barium-coated fecal matter was visible in the rectum and descending colon. After five hours the stomach was empty, and the head of the barium column was in the transverse colon, but it was evident that the sigmoid colon had received the opaque substance from a fistulous tract. A barium sulfate enema confirmed the diagnosis of fistula, the barium going directly from the transverse colon through the fistulous tract into the jejunum. A definite diagnosis of gastrojejunocolic fistula with possible ulceration was made. The roentgenologist added that he did not believe that sufficient barium left the stomach through the pylorus for the patient to get along without another outlet from the stomach when the fistulous tract was resected.

Preparation.—Preoperative treatment consisted in emptying the gastro-intestinal tract of barium and in maintaining a normal level of the body fluids and nutrition. As a portion of the intestinal stream was going directly from the jejunum to the transverse colon through the fistulous tract, it was difficult to empty the cecum and ascending colon. Large doses of liquid petrolatum by mouth and repeated

enemas partially emptied the large intestine, but considerable barium still remained at the time of operation. Because of diarrhea it was deemed inadvisable to attempt a long period of preparation. The patient was given large quantities of fruit juices for only twenty-four hours before the operation, accompanied by a deodor-

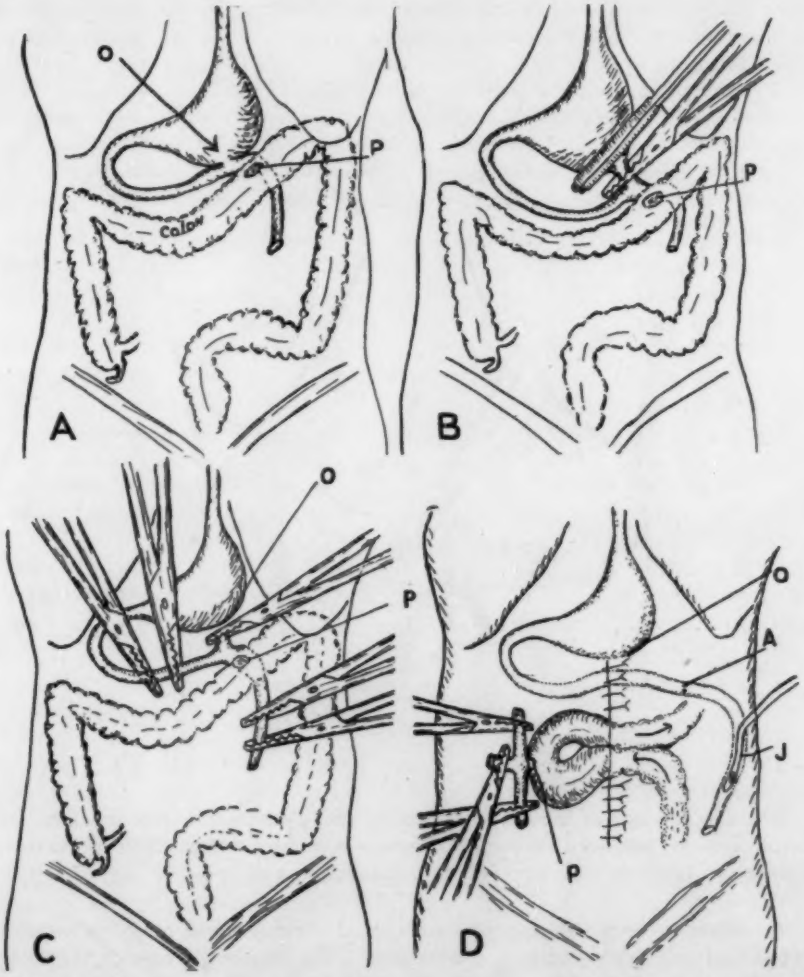

Fig. 1.—In *A* the jejunal ulcer had perforated into the transverse colon on the posterior wall near the mesenteric attachment. *P* indicates the colonic fistula, and *O*, the gastro-enterostomy opening. In *B* the clamps are applied for resection of the gastro-enterostomy. *P* indicates the colonic fistula. In *C* the gastro-enterostomy is separated, and the opening into the stomach is closed. The clamps are in place for resection of the jejunum. *O* indicates the previous opening in the stomach, and *P* the colonic fistula. In *D*, jejunal anastomosis (*A*), completed, with the catheter (*J*) in place for feeding. The loop of transverse colon with the stump of jejunum attached is brought outside of the abdomen for the Mikulicz drain. *P* indicates the colonic fistula.

ized tincture of opium to quiet peristalsis. No enemas were given for the twenty-four hours previous to operation because of the danger of carrying fecal content into the stomach. I preferred to rely on the high acidity of the stomach as a bactericidal agent rather than to depend on a more thorough mechanical cleansing. Nothing was given by mouth for twelve hours prior to the operation. The level of the body fluids was maintained by the administration of dextrose intravenously.

Five hours prior to the operation 100 cc. of a concentrate of amniotic fluid was given intraperitoneally.

Preliminary medication consisted of 6 grains (0.39 Gm.) of sodium pentobarbital and ¼ grain (0.016 Gm.) of morphine given in divided doses. The operation was begun with spinal anesthesia, which was adequate for one hour and twenty minutes. The operation was completed with nitrous oxide anesthesia, supplemented with 2 ounces (60 cc.) of ether, which gave excellent relaxation throughout the entire operation.

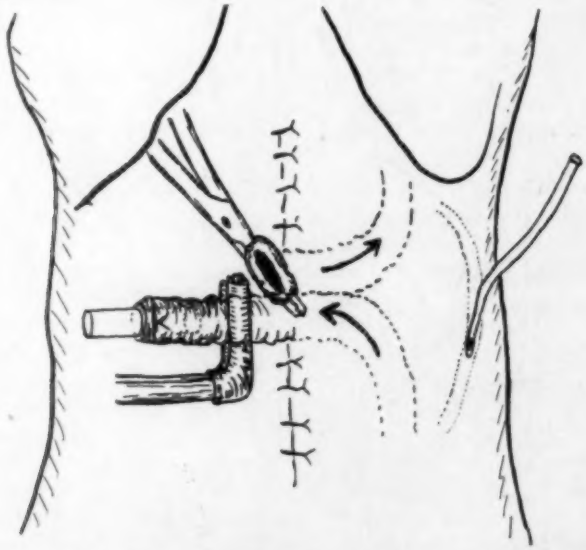

Fig. 2.—The loop of the transverse colon, with the fistulous tract and the stump of the jejunum, has been removed. The tube is in the proximal limb of the transverse colon for fecal drainage (Lahey method).

Operation.—When the abdomen was opened the transverse colon and jejunum were found to be adherent over a wide area. The jejunocolic opening was opposite the gastro-enterostomy stoma. The jejunal ulcer apparently had perforated into the transverse colon on the posterior wall near the mesenteric attachment. Both the gastro-enterostomy and the fistulous opening were large and patulous, readily admitting the thumb. There was surprisingly little gross evidence of recent inflammatory reaction around the fistulous tract, although both the jejunum and the transverse colon showed moderate thickening and injection of the vessels of the intestinal wall.

The stomach presented no gross abnormality, and the only evidence of the original ulcer was a fine white scar on the anterior surface of the duodenum, with two constricting bands, one leading to the gallbladder and the other to the liver.

After these bands were freed the index finger could readily be passed through the pyloric ring and through the first portion of the duodenum.

With some difficulty the adhesions between the stomach, the jejunum and the transverse colon were separated and the fistulous tract was isolated. The gastroenterostomy anastomosis was first severed between clamps, and the opening into the stomach was closed longitudinally without resulting deformity. The jejunum was then divided between clamps placed on each side of the fistulous tract, about 5 inches (13 cm.) of the jejunum being excised. The stump of jejunum, with the fistulous tract leading into the transverse colon, was removed with a wide margin of large intestine. Aseptic closure of the opening into the transverse colon was accomplished.

The severed ends of the jejunum were then united by end-to-end suture. Ten inches (25 cm.) distal to the anastomosis of the small intestine a jejunostomy was

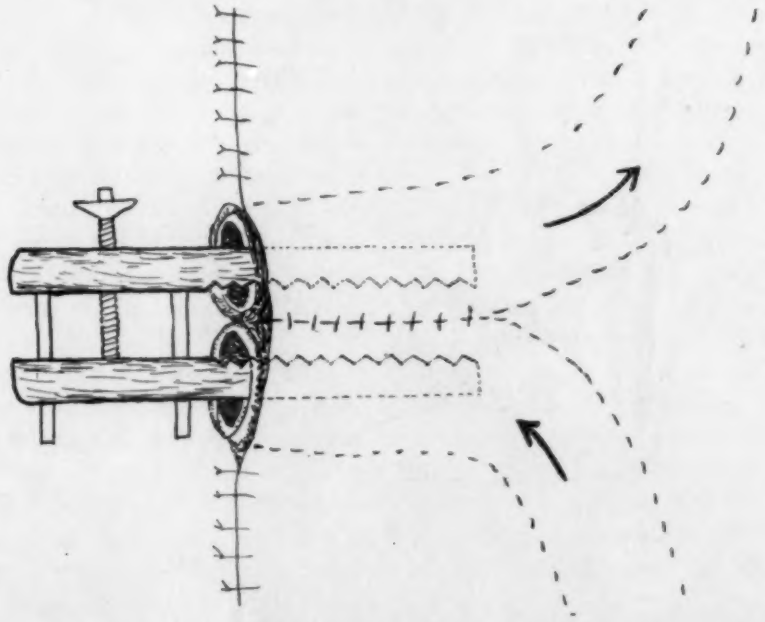

Fig. 3.—The clamp is in place for removal of the septum by pressure necrosis. Note the special teeth on the clamp.

performed by the Witzel method, using a no. 16 double-eyed catheter, which was brought out through a stab wound in the left upper quadrant of the abdomen. This provided a means of feeding the patient until the jejunal anastomosis had healed and would serve for a longer period as well if the duodenum should prove to be as constricted as the roentgenologist had reported. The openings in the mesentery of the transverse colon and into the lesser omental cavity were then closed. To my disappointment, examination then revealed that suture of the transverse colon following excision of the fistulous tract had resulted in marked narrowing of the lumen, with some evidence of impaired circulation in the wall of the bowel. At this time the anesthetist reported that the patient's pulse was slightly irregular, and, as nearly three hours had elapsed since the operation had been started, it seemed imperative to finish quickly. Therefore, the narrow portion of

the transverse colon with questionable circulation was brought outside the peritoneal cavity through the middle of the abdominal incision, according to the method of Mikulicz, and the abdomen was closed.

Postoperative Course.—The postoperative convalescence was very satisfactory. The body fluids and nutrition were maintained at a normal level by the intravenous administration of dextrose for thirty-six hours after the operation. At that time liquids in small amounts were given by mouth, supplemented by feedings through the jejunostomy tube. There was no vomiting, and additions were gradually made to the diet. On the eleventh postoperative day the jejunostomy tube was removed, and the opening closed spontaneously.

Much to my surprise the patient began to pass flatus by rectum on the second postoperative day, and fecal movement began on the fifth day, so that it was

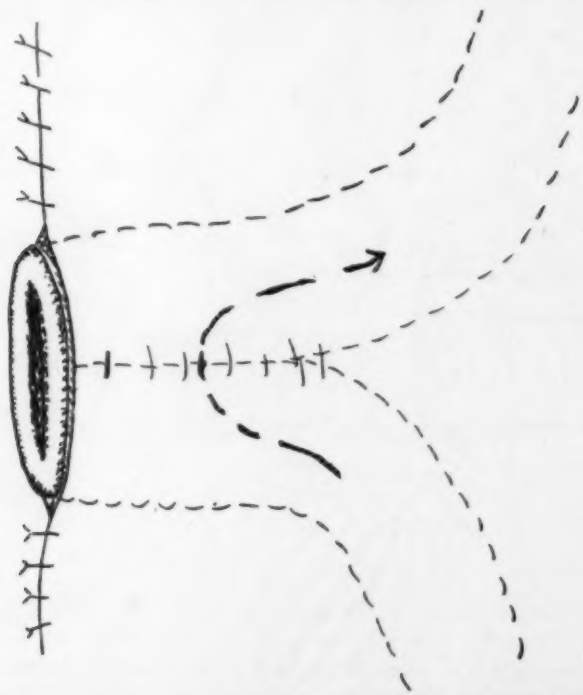

Fig. 4.—The spur is removed, and the fecal stream is restored. Extraperitoneal closure of the colostomy opening can now be performed to complete the procedure by various methods.

unnecessary to open the colostomy stoma for two weeks. At that time the segment of bowel with the constricted lumen was removed with the cautery. The crushing clamp was applied to necrose the septum between the two limbs of the transverse colon. Five days later the clamp came away, and after this most of the fecal stream was passed by rectum. Aside from a superficial infection in the fascia of the abdominal wall, the patient had no complications and was able to leave the hospital on the twentieth postoperative day. He returned three weeks later and closure of the temporary colostomy opening was then done.

At the time of writing the patient's gastric acidity is well controlled by medical measures, and fluoroscopic examination shows no signs of duodenal obstruction or irritation. There are no indications for further surgical intervention. A check-up will be made six months later.[10a]

COMMENT

Walters,[11] of the Mayo Clinic, reported a case of gastrojejunocolic fistula in a debilitated patient with pyloric obstruction on whom he planned to operate in two stages. At the first operation Walters eliminated the gastroenterostomy anastomosis and closed the colonic fistula, performing a jejunostomy to have a means of feeding the patient between operations. Although in his case the roentgenologist had reported no barium leaving the pylorus previous to operation, food passed freely from the stomach immediately after the first operation, and the jejunostomy tube and second operation were never needed. A check-up six months later showed the patient to be in excellent health, symptom-free and with no clinical or roentgenologic evidence of reactivation of his old duodenal ulcer.

Careful search of the literature fails to reveal a previous report of the use of the Mikulicz procedure in cases of this condition. Its advantages in the prevention of peritonitis have been established. Since 60 per cent of the reported deaths in cases of gastrojejunocolic fistula are due to peritonitis, the Mikulicz procedure should be utilized. I am convinced that faced with a similar problem again, I should carry out the Mikulicz procedure immediately after dividing the jejunum and not attempt to excise the fistulous tract at the time of operation. To attempt excision of the fistula and closure of the colon prolongs the operation and needlessly increases the danger of peritonitis. The colonic fistulas in these cases are often so close to the mesentery and so large that any form of suture is likely to result in narrowing of the lumen or impairment of the blood supply.

Involvement of the mesentery of the transverse colon is often observed in cases of carcinoma of the stomach, which requires exten-

10a. The patient gained 40 pounds (18 Kg.) in three months. He returned six months after the operation for fascia lata repair of the inguinal hernia. At that time he was free from symptoms, and the gastric analysis showed: free hydrochloric acid 46 and total acid 58. The patient was able to do his usual work as a rancher. A roentgenogram made one year after the operation failed to show any evidence of duodenal ulcer; the stomach was entirely empty of barium sulfate in two hours. The patient's acidity is controlled by frequent feedings and by the omission of tobacco. Gastric analysis showed free hydrochloric acid 50 and total acid 60, one year after the operation.

11. Walters, Waltman, and Priestley, J. T.: Proc. Staff Meet., Mayo Clin. **8**:33 (Jan. 18); 339 (May 31) 1933.

sive block dissection if any attempt is made to treat the lesion radically. The Mikulicz procedure should be of value here as well as in cases of gastrojejunocolic fistula.

SUMMARY

A brief review of the etiology, altered physiology and present methods of dealing with gastrojejunal ulcer and its complications is given.

A case of gastrojejunocolic fistula with successful treatment by graded operation is reported.

The additional safety factors of preoperative peritoneal immunization, simplified multiple stage operations and the use of the Mikulicz procedure are stressed.

HEPATIC TRAUMA

ANALYSIS OF SIXTY CASES

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DETROIT

This paper is a survey of the cases of sixty patients who had undergone hepatic trauma who were brought to the City of Detroit Receiving Hospital during the period from 1927 to 1934, inclusive, and represents with some degree of accuracy the frequency of this condition in Detroit. Practically all patients with a condition of this nature are brought into the emergency ward by police ambulance a short time after the accident.

The distribution of the patients in regard to age, color and sex is shown in table 1. The majority were in the so-called age period of greatest activity.

TABLE 1.—*Age, Color and Sex Distribution of Patients*

Age, Years						Color and Sex			
						Negro		White	
3 to 4	10 to 20	20 to 30	30 to 40	40 to 50	Over 50	Male	Female	Male	Female
2	9	29	10	7	3	21	4	31	4

TABLE 2.—*Mode of Injury*

Gunshot (41)												Trauma (10)				Stab (9)	
Quarrel		Police		Robbery		Accident		Suicide		Unclassified		Automobile		Fall			
Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female	Male	Female
9	7	6	0	6	0	5	0	1	0	8	0	7	2	1	0	7	2

Injury to the liver in every case in this series was caused by external violence. The patients were divided into three groups: those with gunshot wounds, 68 per cent, those with trauma, such as that caused by an automobile, a street-car or a fall, 17 per cent, and those with stab wounds, 15 per cent. The largest subgroup in any of these divisions was made up of persons taking part in the eternal triangle, and, as one can surmise, about 50 per cent of the patients were women (table 2).

OBSERVATIONS

Symptoms on Admission.—The most common symptom on the patient's admission to the hospital was shock, with its attending signs, such as pallor, cold, clammy skin, restlessness, rapid, thready pulse, subnormal temperature, thirst and, at times, vomiting. The degree of shock varied roughly in direct proportion to the extent or the seriousness of the trauma and in the majority of the cases indicated the extent of coexisting hemorrhage. In 73 per cent of the cases signs of shock were present or reappeared after treatment and continued until death.

The degree of pain and its distribution depended on the extent and the character of the injury. In a few cases of clean stab wounds the pain was so slight that the patient walked to the hospital. The extensive or macerating injury produced the most pain.

None of our patients showed bradycardia, as described by McKnight [1] and by Erdély,[2] but when this symptom is present, one must not underestimate the degree of hemorrhage.

Blood Counts on Admission.—Laboratory evidence of anemia and intra-abdominal injury was present at the time of admission in the twenty-four cases in which complete blood counts were made. In all but two of these cases there were severe intra-abdominal hemorrhages.

The hemoglobin content (Dare) varied from 40 to 90 per cent and in one half of the cases it was below 70 per cent. The red cell count varied from 2,800,000 to 5,500,000. In 77 per cent of the cases the count was below 4,000,000. The white cell count varied from normal to 33,000, and in 62 per cent of the cases it was above 10,000. The polymorphonuclear count varied more constantly than any other finding and ranged from a normal value to 90 per cent. In 91 per cent of the cases the count was over 70 per cent, and in 71 per cent it was over 80 per cent. Complete blood counts, and especially the red cell count and the hemoglobin content, are valuable aids in differentiating hemorrhagic shock and traumatic shock; they are of still greater value when determinations are repeated at regular intervals for comparison.

Operation.—Fifty-five patients were operated on; five were treated medically. Operation was performed promptly after adequate response to the treatment for shock and hemorrhage.

Ether was used in forty-four cases; spinal analgesia, in twelve cases. In view of a previous survey [3] on the use of spinal analgesia in emer-

1. McKnight, R. B.: Post-Operative Physiological Studies in a Case of Traumatic Liver Rupture, Am. J. Surg. **8**:542 (March) 1930.

2. Erdély, J.: Recovery from Multiple Ruptures of the Liver with Intestinal Injury: Diagnosis and Treatment of Subcutaneous Rupture of the Liver, Deutsche Ztschr. f. Chir. **198**:110, 1926.

3. Krieg, E. G.: Spinal Anaesthesia in Cases of Emergency Surgery, J. Michigan M. Soc. **31**:456 (July) 1932.

gency cases, I do not believe that it should be used in the presence of severe hemorrhage such as one finds in cases of hepatic trauma.

The hepatic wound was treated by packing in forty-one cases and by suture in fifteen cases. It is of interest to note that in the majority of cases the gauze was not inserted into the wound with enough pressure to control hemorrhage and frequently it was merely placed against the wound, yet in no case was there alarming bleeding after the patient returned to the ward. In several of the cases large amounts of gauze were packed into an area of extensive trauma, and in all these there developed symptoms of obstruction. Dragstedt [4] has shown in dogs that even a minor degree of pressure in this region easily produces obstruction. Walters [5] has shown experimentally and clinically that a definite syndrome is set up by pressure about the liver.

The experiences of Anderson,[6] Halstead,[7] Robertson and R. R. Graham,[8] A. J. Graham [9] and Tilton [9a] suggest that the insertion of a small pack or drain into the deep hepatic wounds is the method of choice. Drainage is imperative for deep hepatic wounds, to prevent encapsulated collections of blood, bile and pus. Suture of the clean superficial wound is adequate. Leakage of bile in some degree is present in all cases, and it would appear that abdominal drainage of some type is necessary regardless of the technic used.

Treatment Without Operation.—Of the five patients treated medically, two died within three hours. The other three were children in an extreme state of shock, and improvement was not shown until after the third day.

One patient had a gunshot wound through the dome of the liver and the diaphragm, and signs of subdiaphragmatic abscess developed six weeks later. Transpleural drainage was done, but no pus was obtained. The wound drained for several days and healed promptly. The patient was discharged two weeks later. It is most probable that her condition was similar to that in the cases described by Robertson and Graham,[8] Graham [9] and Anderson,[6] in which a collection of blood or bile was present in the substance of the liver.

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4. Dragstedt, C. C.: Acute Dilatation of Stomach, J. A. M. A. **79**:612 (Aug. 19) 1922.

5. Walters, W.: Accumulated Bile Displacing Liver, Surg., Gynec. & Obst. **47**:421, 1928.

6. Anderson, L.: Central Ruptures of Liver and Their Complications, Beitr. z. klin. Chir. **135**:696, 1926.

7. Halstead, A. E.: Four Cases Requiring Liver Surgery, S. Clin. North America **3**:973 (Aug.) 1923.

8. Robertson, D. E., and Graham, R. R.: Rupture of Liver Without Tear of the Capsule, Ann. Surg. **98**:899 (Nov.) 1933.

9. Graham, A. J.: Subcutaneous Rupture of the Liver, Ann. Surg. **86**:51 (July) 1927.

9a. Tilton, B. J.: Considerations Regarding Wounds of Liver, Ann. Surg. **9**: 27 (Jan.) 1905.

Another patient, who had been struck by an automobile, was in poor condition for four days and then gradually improved. All signs of intra-abdominal hemorrhage were present, including a fluid wave in the abdomen. The hemoglobin content was 40 per cent, and there were 2,950,000 red blood cells. He was discharged on the twenty-third day.

The third patient, who had been run over by an automobile, improved under treatment for a week. On the eighth day restlessness and abdominal pain began, and they continued for four days. On the tenth day abdominal distention began, continuing until death, and the pulse rate, which had ranged within normal limits, began to rise slowly. The patient became irrational on the fifteenth day, and vomiting began on the seventeenth day. Death occurred nineteen days after the accident.

At autopsy the liver revealed a large healed laceration, with fibrosis in the right lobe near the gallbladder. The gallbladder was ruptured, and the abdomen was filled with bile.

Progress After Primary Shock or Operation.—In all the patients who survived the effect of shock and hemorrhage the condition progressed in one of two ways. The recovery of the first group was comparable to that of any other group of patients on whom laparotomy has been done for a "clean" condition. In the second group certain symptoms developed, and there were complications which were attributed to the pathologic condition of the liver. In this group there were thirty-one patients.

Irrationality or stupor was noted in thirteen cases. This symptom was more frequently intermittent but at times it was continuous; it was noted mostly at night. Extreme irritability was noted in seven cases. The characteristics of this symptom paralleled those of irrationality. It is probable that this symptom is only a stage of irrationality. Vomiting was present in eleven cases and suggested that found when the stomach is dilated or obstructed. Jaundice was present in one patient; it appeared in the first twenty-four hours.

The onset of these symptoms was more frequent during the first three days and especially on the second day. None of our patients survived in whom any one of these symptoms persisted for two days or longer. None lived when any combination of these symptoms appeared. The mortality of the combined group of patients exhibiting the first two symptoms was 85 per cent. The total mortality for the entire group was 74 per cent (tables 3 and 4).

The pulse rate averaged about 90 per minute, and the average temperature was about 99 F., except for the postoperative and the preagonal rise. These observations certainly point to a toxic condition on a noninfective basis.

The third of the patients treated without operation whose cases have been described had all the symptoms and findings under discussion, and his case supports the contention that the condition was toxic without infection.

Bronchitis was present in six cases, but all the patients recovered rapidly. Abdominal distention requiring active treatment occurred in five cases, one of which was an instance of infective peritonitis following secondary closure.

Hepatic Wounds.—Hepatic wounds are divided generally into deep and superficial wounds. Four types are further described in this survey, as follows: (1) clean incision produced by a stab, (2) clean puncture by a bullet, (3) macerating puncture by a bullet and (4) rough fracture caused by contusion.

TABLE 3.—*Onset of Symptoms (in Days)*

	1	2	3	4	6	8	9	14	15	19	Total
1. Irrationality	2	5	2	..	1	..	1	1	..	..	13
2. Irritability	1	4	1	..	..	1	..	..	..	1	7
3. Vomiting	1	4	2	1	1	1	..	..	1	1	11
Total	4	13	5	1	2	2	1	1	1	2	31

TABLE 4.—*Duration of Symptoms in Days*

	1	2	3	4	5	14	Total
1. Irrationality	5	3	2	2	1	..	13
2. Irritability	4	1	1	..	1	..	7
3. Vomiting	5	4	..	..	1	1	11
Total	14	8	3	2	3	1	31

TABLE 5.—*Mortality in Relation to the Lobe Injured*

	Right Lobe			Left Lobe		
Type of Injury	No. of Patients	Died	Lived	No. of Patients	Died	Lived
Stab	5	3	2	4	0	4
Trauma	8	7	1	2	2	0
Bullet, clean wound	9	5	4	6	3	3
Bullet, macerating wound	17	9	8	7	6	1
Total	39	24	15	19	11	8

The right lobe was involved in thirty-nine cases, the left lobe in nineteen and both lobes in two. In two thirds of all the fatal cases there was injury to the right lobe (table 5).

In forty-one cases the liver was the only organ involved. In the cases of injury to the right lobe there was higher mortality. The deep wounds caused all the deaths; the seven patients suffering from superficial wounds lived even when severe hemorrhage was present (table 6).

Other Organs Involved.—In twenty-one cases other organs were involved; in all these the injury was caused by gunshot, except in one,

in which an automobile accident caused rupture of the gallbladder. The spleen was involved in more cases than was any other organ, and damage to it was always associated with injury to the left lobe of the liver. The mortality in this group was 94 per cent (table 7).

Hemorrhage.—Abdominal hemorrhage was found in every case and varied in degree, depending on the extent of the injury. The extent and the severity of the injury coincided in the majority of cases. Hem-

TABLE 6.—*Data on Forty-One Cases of Hepatic Damage Only*

	Stab				Trauma				Clean Bullet Wound				Macerating Bullet Wound			
	Right Lobe		Left Lobe		Right Lobe		Left Lobe		Right Lobe		Left Lobe		Right Lobe		Left Lobe	
Degree of Injury	L	D	L	D	L	D	L	D	L	D	L	D	L	D	L	D
Deep	2	3	4	0	1	6	2	0	1	3	2	1	4	4	1	0
Superficial	..	..	..	..	..	..	..	..	3	0	..	..	4	0	..	..

TABLE 7.—*Other Organs Than Liver Involved, Twenty-One Cases*

	Right Lobe	Left Lobe	Both Lobes	Total	Recovery	Death
Spleen	0	4	0	4	0	4
Stomach	2	3	1	6	0	6
Small bowel	2	1	0	3	1	2
Diaphragm	3	1	0	4	0	4
Colon	1	1	0	2	0	2
Spinal section	1	0	0	1	0	1
Gallbladder	1	0	0	1	0	1
Total	10	10	1	21	1	20

TABLE 8.—*Mortality in Relation to the Degree of Abdominal Hemorrhage*

Degree of Hemorrhage	Death	Recovery
Slight	0	2
Moderate	2	7
Severe	35	14

orrhage was classified as slight, moderate and severe. Tables 8 and 9 show interesting findings.

The more recent conception of the mechanism of shock produced by hemorrhage and its treatment has been clearly described by Freeman,[10] in a recent paper. Loss of large amounts of blood causes a powerful stimulation of the adrenal glands to produce epinephrine, in an effort to keep up an adequate circulation of fluid by constriction of the peripheral

10. Freeman, N. E.: Hemorrhage in Relation to Shock, Ann. Surg. **101**: 484 (Jan.) 1935.

vessels. Repeated or continuous hemorrhage continues the stimulation until a point is reached after which no therapy is successful. Early replacement of an adequate amount of fluid is necessary and promptly gives good results. His experiments pointed out that transfusion of blood or administration of a 6 per cent solution of acacia gives the best results. This work also contraindicated the giving of medicinal stimulants, especially epinephrine.

TABLE 9.—*Hemorrhage in Relation to the Degree of Injury*

	Slight	Moderate	Severe	Death	Recovery
Stab	0	2	7	3	6
Trauma	0	0	10	9	1
Clean bullet wounds	1	4	10	8	7
Macerated bullet wounds	1	3	22	17	9
Superficial bullet wounds	1	2	4	0	7

TABLE 10.—*Mortality Caused by Various Agents*

Agent	Died	Lived
Gunshot	25	16
Stab	3	6
Trauma		
Automobile	8	1
Fall	1	..
Total	37	23

TABLE 11.—*Complications Causing Death*

	24 Hrs.	48 Hrs.	72 Hrs.	4 Days	5 Days	6 Days	7 Days	8 Days	9 Days	10 Days	21 Days
Hemorrhage and shock	20	3	4	1	..	..	..	..	..	..	..
Toxic (bile) peritonitis	..	..	..	..	1	3	..	1	..	1	1
Infective peritonitis	..	..	..	..	..	..	1	..	..	..	..
Mediastinal pressure	..	..	..	..	..	..	..	..	1	..	..
Total	20	3	4	1	1	3	1	1	1	1	1

Mortality and Cause of Death.—The mortality in the entire series was 61.6 per cent; the mortality in cases in which operation was performed was 56.6 per cent, and that in cases in which there was no operation was 60 per cent. Table 10 shows the relation between the agent of trauma and the mortality.

Hemorrhage and shock caused death in 73 per cent of the cases in which they occurred, and all the patients died within three days, only a few living longer than twenty-four hours. Because all these cases were "coroner's cases," autopsy was performed by us in only three. A number of deaths termed "toxic deaths" probably belong to the "bile peritonitis" group, because they resembled them clinically in all details (table 11).

SUMMARY

The mortality for the whole group was 61.6 per cent; the mortality in the group in which operation was performed was 56.6 per cent. The most serious prognosis obtains in cases of contusion (caused by automobile accident, etc.), deep wounds or injury to the right lobe of the liver and in those in which contiguous organs are also involved.

Irrationality, irritability, vomiting and jaundice are serious prognostic signs.

Hemorrhage was always present, extensively in 80 per cent of the cases. Immediate adequate replacement of fluid is indicated; epinephrine should not be given.

Deep hepatic wounds are best treated by small drains inserted deeply; suture is adequate for superficial wounds; abdominal drainage is necessary in all cases.

Shock due to trauma and hemorrhage caused 73 per cent of the patients in whom they occurred to die within seventy-two hours. Bile peritonitis was the next most frequent cause of death.

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